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Practical Electrocardiography

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Illustrated by MARCIE ETHRIDGE PERRY

SECOND EDITION



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For he who'd make his fellow creatures wise
Should always gild the philosophic pill'

W S GILBERT

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Preface to Second Edition

The aims and scope of this book are unchanged and simplicity remains the central theme The text is designed to be digestible for beginners yet not without value to those who already have a nodding acquaintance with electrocardiography

The whole text has been meticulously revised and new sections have been included on the following subjects the T_P wave the U wave relation of standard to unipolar limb leads systolic and diastolic overloading of the ventricles parasystole coronary sinus rhythm main stem extrasystoles differentiation between ventricular and supraventricular tachycardias intra atrial block nodal and ventricular escape A V dissociation with interference infarction without Q wave electrocardiographogenic di ease post extrasystolic T wave changes hyperkalemia and hypocalcemia

A number of selected references have been included and these have been chosen with an eye not only to their value but also to their accessibility After each of the last six chapters review tracings without descriptive legends have been inserted to serve as a cursory refresher course and as a stimulus to master what has gone before

Seventy six new illustrations have been added while 26 of the old ones have been eliminated Once again the publishers have graciously given me a free and unconventional hand in arranging the layout of pages so that I have been able to place illustrations and descriptive text in as convenient proximity as possible

H J L M

Preface to First Edition

Books on electrocardiography seem to possess one or more of several disadvantages for the beginner: the introductory chapters on electrophysiology are so intricate and longwinded that the reader's interest is early drowned in a troubled sea of vectors, axes and gradients; or only certain aspects of the subject are dealt with, for example the arrhythmias may be entirely omitted; or illustrations are deficient and frequently situated uncomfortably far from the descriptive text.

For several years I have been attempting to introduce fourth year students to the comparatively easy technique of interpreting electrocardiograms. During this period I have been unable to recommend any single text that deals with the subject quickly and simply and yet is sufficiently comprehensive. This book is an attempt to supply such a manual. Its aims are: 1) to emphasize the simplicities rather than the complexities of the electrocardiogram; 2) to give the reader only those electrophysiologic concepts that make everyday interpretation more intelligible without burdening him with unnecessary detail; 3) to cover all diagnostically important electrocardiographic patterns; and 4) to provide adequate illustrations and in every instance to have the illustration conveniently situated to the reader as he reads the descriptive text. To achieve this last desideratum the publishers have generously waived publishing conventions and given me a free hand in the arrangement and spacing of illustrations and text.

This book is designed for those approaching electrocardiography from the point of view of the clinician. It is hoped that it will enable the beginner to acquire a rapid but thorough grasp of a sophisticated yet simple discipline.

H. J. L. M.

ACKNOWLEDGEMENTS

It is a great pleasure to acknowledge my indebtedness

To the electrocardiograph department at Mercy Hospital for so freely placing their files at my merciless disposal

To Dr William Schuman for coining the *mot juste* electrocardiographogenic

To the publishers whose gracious cooperation has been constant and unfailing especially to Mr Dick M Hoover who has devoted many hours to assisting me with the layout of pages so as to achieve optimal coordination of illustrations with text

H J L M

Contents

Introductory Note

Electrocardiography is generally simple but this does not mean that it always provides a clearcut answer The electrocardiogram has definite limitations which must be thoroughly appreciated In the arrhythmias and blocks it usually gives a specific and irrefutable answer but with myocardial disease there is much less specificity Every interpreter no matter how experienced at times encounters tracings which he cannot unravel Not infrequently a tracing is borderline or abnormal but non specific and must be classified as such—an unsatisfying situation for both the interpreter and the clinician in charge of the case yet one which must be frankly and humbly faced

Profession and laity alike are inclined to lay too much stress on mechanical devices in diagnosis The electrocardiogram which is far from infallible is no exception It should be remembered that a patient with a normal tracing may drop dead of a coronary attack five minutes later while another with a grossly abnormal tracing may live on without cardiac symptoms for many years

It cannot be too emphatically urged that the electrocardiogram should always be read in the clearest possible light of clinical observation All pertinent clinical data hould be in the hands of the interpreter Ideally the clinician in charge of the case reads his own tracings failing this he should ee to it that his interpreting colleague is furnished with full clinical details including his own clinical impression for only so will he and his patient derive maximal benefit from expert interpretation

As electrocardiographic interpretation and clinical observation are or should be inseparable a certain number of clinical notes of practical diagnostic value are included in this primarily electrocardiographic text

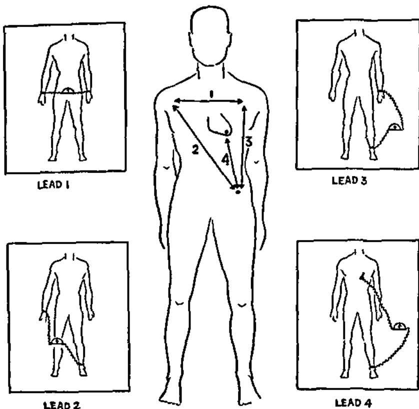


FIG 1 The standard limb leads and the first precordial lead

Caution The doul le headed arrow in the diagram are not intended to represent Einthoven s triangle

1

Electrodes and Leads

STANDARD LIMB LEADS

These are three in number and have been in use for about fifty years We therefore have far more empirical knowledge of these leads than of the numerous additional leads more recently introduced It is probably true to say that about eighty to ninety per cent accuracy in diagnosis can be achieved by inspecting these leads alone Most arrhythmias and most types of heart block are easily diagnosed from these leads alone

The connections of these leads are illustrated in figure 1 Lead 1 connects the two arms lead 3 connects the left arm with the left leg while lead 2 the hypotenuse of the triangle connects the right arm with the left leg Each lead records the difference in potential between the two connected limbs Although the electrodes are attached at wrists and ankle this is purely a matter of convenience—it is easiest to attach bracelets to these parts of the limbs It is more accurate to think of the potential as derived from the roots of the respective limbs i e from the two shoulders and the left groin The heart is approximately in the center of the triangle so formed (fig 1)

It is worth noting **Einthoven s law** which states in effect that a complex in lead 2 is equal to the sum of the corresponding complexes in leads 1 and 3 (2 = 1 + 3) This is a helpful rule to remember when the technician has wrongly labelled the leads For example if the P wave is seen to be upright in all three leads you know at a glance that the lead with the tallest P is lead 2

PRECORDIAL LEADS

The standard limb leads have two disadvantages 1) each is derived from *two* points *distant* from the heart and 2) the three electrodes are all in the same plane i e the frontal plane of the body It is not surprising that additional information can be gained by placing electrodes closer to the heart and moving such electrodes round the bend of the thorax to obtain views of the heart from different angles The first precordial or chest lead introduced in 1932 connected the left leg with the apex beat and was called lead 4 (fig 1) This was a successful innovation and soon a series of precordial points was introduced whose positions are illustrated in figure 2 Point 1 is just to the right of the sternum in the fourth interspace point 2 just to the left of the sternum in the fourth interspace Point 4 lies in the midclavicular line in the fifth interspace Point 3 is halfway between 2 and 4 Points 5 6 and 7 are at the same level as 4 in the anterior middle and posterior axillary lines respectively

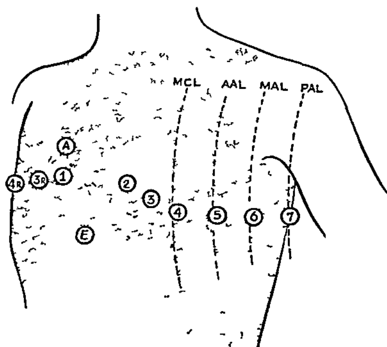


Fig 2 Precordial points from which chest leads are derived

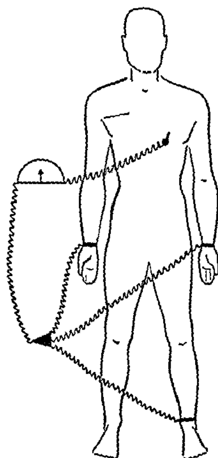
Most cardiologists employ precordial positions from 1 to 6 routinely Sometimes it is desirable to take additional leads from further points some of these are also illustrated in figure 2 Point E is situated over the ensiform process The atrial lead point (A) is just to the right of the sternum in the third interspace The locations of further right precordial points 3_R 4_R etc correspond with the locations of their opposite numbers on the left side of the chest Occasionally it is desirable to take leads from the posterior thorax points 8 and 9 are at the angle of the scapula and over the spine at the same level as 4 5 6 and 7

It is convenient though not entirely accurate to think of these precordial points as picking up potentials from certain areas or chambers of the heart A lead derived from the atrial lead point is thought of as a right atrial lead (If a left atrial lead is required an esophageal electrode is used) Leads derived from points 1 2 E 3_R and 4_R are conveniently thought of as right ventricular leads Leads from points 5 6 and 7 are regarded as left ventricular leads while transitional leads are derived from 3 and 4 and are most likely to reflect septal currents It should be emphasized that this is rough geography but holds true for the majority of hearts Changes in heart position or in the relative size of the ventricles may materially alter these relationships In fact as will be seen in Chapter 5 right and left ventricular and transitional leads are better recognized by their configuration than by their precordial points of origin For a note on the use of the terms left ventricular lead etc see page 48

In a standard lead the two electrodes are about equally remote from the heart and are therefore about equally important in their contribution to the tracing When however one electrode is placed in one of the precordial positions while the other electrode is on a limb it is natural that the closer chest electrode should contribute more to the tracing and the limb electrode less The limb attachment of such a lead is therefore called the **indifferent electrode** while the chest electrode is referred to as the **exploring electrode** since it is moved in an exploratory fashion from point to point across the chest If the indifferent electrode is attached to the left leg the connection is designated CF if to the right arm CR if to the left arm CL According to the precordial point employed a subscribed number is added to the CF CR or CL label Thus for example lead CF_5 indicates that the exploring electrode is placed at point 5 in the anterior axillary line while the indifferent electrode is attached to the left ankle

V LEADS

Though the exploring electrode exerts a far greater influence on the tracing than the indifferent electrode the indifferent electrode nevertheless has considerable influence It was discovered empirically however that if all three limb electrodes were connected through resistances of 5000 ohms each to form a common **central terminal** this afforded a more truly indifferent connection For it was shown that the potential at such a central terminal was practically zero throughout the cardiac cycle Thus theoretically at any rate such a connection leaves the exploring electrode as sole dictator of the pattern The hookup of the V leads is diagrammatically shown in figure 3



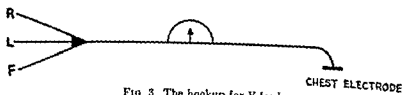


FIG 3 The hookup for V leads

Although theoretically the V lead connections should give the most reliable precordial pattern in practice it is not so certain that they always do (2) and it is worth appreciating the expected differences between the various precordial connections The e may best be summarized by stating that the CR leads tend to empha ize positive (upright) deflections while the CF leads lend emphasis to negative (downward) waves The pattern of V leads usually lies somewhere in between In other words it may sometimes be useful to take advantage of the emphasizing tendencies of the CR and CF connections, rather than to rely slavishly on the V leads because they are theoretically superior Such employment of the CR or CF leads may be likened to the use of a magnifying glass to detect otherwise invisible or questionable changes Figure 4 illustrates the difference between CF and V tracings taken from identical precordial positions The difference in respective T waves is readily apparent This figure will again be referred to in the discussion of coronary disease

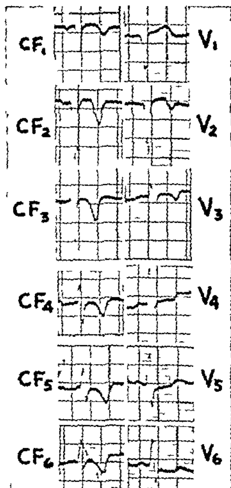
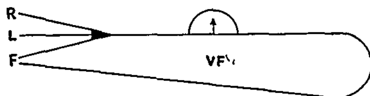


Fig 4 Comparison of CF and V tracings on the same patient taken from identical precordial points

aV LEADS

The standard limb leads are strictly **bipolar** representing as they do the difference in potential between two points CF CR and CL leads are also clearly bipolar in that they too record the difference in potential between two points In the standard leads the two points involved exert approximately equal influences whereas in the C leads as stated above the precordial point is more influential than the distant limb connection With the V leads comes the virtual exclusion of this distant influence because the central terminal shows practically zero potential throughout the heart cycle They are therefore referred to as **unipolar** precordial leads From this development it is only a short step to the unipolar *limb* leads By using the central terminal as the indifferent connection and placing the exploring elec



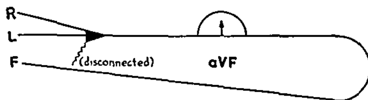


Fig 5 A The hookup for lead VF B The hookup for augmented VF (aVF)

trode on one limb the resulting tracing might well be expected to record the potential at the root of the explored limb exclusively. Such leads are labeled VR, VL and VF according to the limb with which the exploring electrode is connected. The connections of VF are diagrammatically shown in figure 5 A. The deflections in such a lead are small.

It was soon empirically discovered that the amplitude of complexes in such leads could be materially increased by disconnecting the central terminal attachment to the explored limb (fig. 5 B). This device increased the size of the deflections (thus making them more readable) without significantly altering their shape. This *augmentation* of potential is designated by a prefixed a —aVR, aVL, aVF.

RELATION OF BIPOLAR TO UNIPOLAR LIMB LEADS

It is therefore apparent that, in a sense, the unipolar limb leads are the algebraic bricks of which the bipolar leads are built. Lead 1 clearly represents the difference between aVR and aVL, lead 2 the difference between aVR and aVF, and lead 3 between aVL and aVF.

Now, quite arbitrarily, as originally ordained by Einthoven, the polarity of the electrodes is arranged as in figure 6. F is positive in relation to R in lead 2 and relative to L in lead 3, while in lead 1 L is positive in relation to R. In other words F is always relatively positive and R is always relatively negative, while L is variable as between leads 1 and 3. The relationships between the bipolar and unipolar limb leads can thus be summarized in the equations

lead 1 = aVL − aVR
lead 2 = aVF − aVR
lead 3 = aVF − aVL

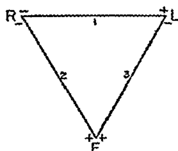


Fig 6. Polarity of electrodes in the standard leads

Theoretically the information available from the six limb leads can be deduced from any two of them In theory therefore it is only necessary to take two of the limb lead but in practice it is probably more valuable to acquire a working knowledge of all six If only two leads are taken it has been found that the most suitable and informative pair are 1 and aVF (5)

SUMMARY

It is generally accepted that in a routine screening electrocardiogram twelve leads should be employed the three standard limb leads the three aV leads and six V leads from 1 to 6 inclusive It should be understood however that for certain purposes this number of leads is inadequate while for other purposes twelve lead are quite unnecessary In following the progress of an arrhythmia a single lead 2 or 3 is usually ample whereas in a doubtful case of myocardial infarction it may be wise to employ CF as well as V connections and to explore additional higher and more lateral areas of the precordium There should be no rigid routine While the usual twelve leads are generally adequate and necessary the number should be freely modified or supplemented by an intelligent understanding of the particular requirements

REFERENCES

1 Douglas A H and Cohen V The vector and algebraic relationship of the CF and V chest lead Am Heart J 1954 48 40
2 Editorial Are the V leads always superior? Ann Int Med 1952 36 1548
3 Schaffer A I et al A new look at electrocardiographic leads Am Heart J 1956 52 04

2

Rhythm and Rate

In every electrocardiogram nine features should be examined systematically

1. Rhythm
2. Rate
3. P wave
4. P R interval
5. QRS complex and interval
6. ST segment
7. T wave
8. U wave
9. Q T duration

A suggested form for recording routine interpretations is given on page 13

RHYTHM

A glance is enough to determine whether the rhythm is regular or irregular If it is regular the interpreter should state whether it is sinu atrial (S A)—as it usually is—A V nodal or idioventricular If it is irregular a preliminary survey should be made to determine whether there is a definite pattern to the irregularity e g beats grouped in pairs every fourth beat dropped etc or whether the irregularity is erratic as in atrial fibrillation

TIMING

The tracing is inscribed against a background of millimeter squares and every fifth line is thicker than the intervening four The horizontal span between two consecutive thick lines is ⅕ second (0 2 sec) the time elapsing between two consecutive thin lines is $\frac{1}{25}$ second (0 04 sec) The basic interval for timing electrocardiographic events is thus 0 04 seconds In practice, if an interval is to be measured one counts the number of small squares horizontally contained within the interval and multiplies this number by 0 04 It is an easy matter to multiply by 4 and adjust the decimal point In figure 7 it can be seen that about two squares are horizontally contained between the beginning and the end of the QRS complex the QRS interval in this case is therefore 2 × 0 04 = 0 08 sec The P R interval (from beginning of the P to the beginning of the QRS) measures 3½ small squares and is therefore 0 14 sec

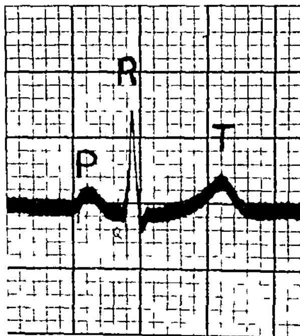


Fig Measurement of P R and QRS intervals

SUGGESTED FORM FOR ROUTINE RECORDS

RHYTHM RATE

P R INTERVAL QRS INTERVAL Q T DURATION

P WAVE

QRS COMPLEX

axis

electrical position

transitional zone

Q waves

ST SEGMENT

T WAVE

U WAVE

IMPRESSION

To determine the rate of the heart we use mainly the thick lines as points of reference As there are 300 fifths of a second in a minute (5 × 60) it is only necessary to determine the number of fifths of a second between consecutive beats (if the rhythm is regular) and divide this number into 300 For convenience we select a complex which coincides with a thick line and then count the number of fifths elapsing before the same complex recurs The QRS complex is usually employed but it is obvious that any wave will serve provided the rhythm is normal and regular Should there be only 1/5 second between consecutive beats the rate will be 300 if 2/5 150 if 3/5 100 and so on Table 1 gives the rates prevailing if from one to ten fifths elapse between consecutive beats For rates between 30 and 100 it is obvious that reasonably accurate approximations can be made at a glance

In the second example (b) in figure 8 the QRS marked x coincides with a thick line There are then 6½ fifths of a second (thick lines) before the next QRS is reached Thus the rate will obviously lie about halfway between 50 and 43 and may be called 46 or 47 with conviction that the approximation is within 1 or 2 beats of the actual rate This method is obviously quite accurate enough for all practical purposes and the slower the rate the more accurate the approximation For even greater accuracy the intermediate figures provided in the guide in figure 9 may be employed (These figures are obtained by dividing into 1500 the number of 25ths of a second elapsing between consecutive beats)

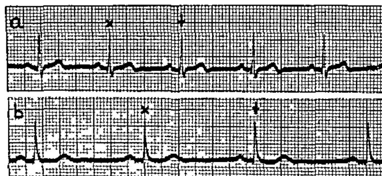


FIG 8 Estimation of heart rate (two examples) See text and fig 9

TABLE 1

WITH THIS NUMBER OF FIFTHS BETWEEN CONSECUTIVE QRS COMPLEXES	THE RATE IS
1	300
2	150
3	100
4	75
5	60
6	50
7	43
8	37
9	33
10	30

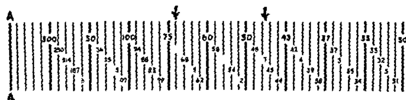


FIG 9 Guide for rapid estimation of heart rate In any tracing select a QRS complex that coincides with a thick line (e.g. those marked x in the two examples in fig 8) This thick line is represented by the first thick line AA in the guide above Note with which line the next QRS in the tracing coincides (arrows in fig 8) and read off the rate from the corresponding line of the guide First arrow rate 71 (tracing a) second arrow rate 46 (tracing b) NOTE UNLIKE MANY MANUFACTURERS RULERS THIS GUIDE IS NOT DRAWN ON THE SAME SCALE AS THE CLINICAL TRACING AND IS OBVIOUSLY NOT INTENDED FOR DIRECT APPLICATION TO IT IT IS A DIAGRAM REPRESENTING THE LINES IN THE TRACING AND IS INTENDED AS AN AID TO MEMORIZING KEY FIGURES THAT EXPERIENCED INTERPRETERS CARRY IN THEIR HEADS

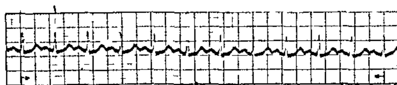


FIG 10 Estimation of rapid rates In the 5 seconds between the two markers there are 11 cardiac cycles The rate is therefore 11 × 12 = 132 per minute

When the rate is over 100 the margin of error rapidly increases and to determine the rate more accurately, it is better to count the number of cardiac cycles occurring in 5 6 or 10 seconds and multiply the number by 12 10 or 6 This method must also be adopted regardless of the rate when the rhythm is irregular Figure 10 illustrates this method for estimating a rapid rate

Some electrocardiographic paper (e g Sanborn) conveniently provides marginal markers at 3 second intervals On such records the simplest and quickest method for estimating rate is to count the number of cardiac cycles in six seconds and multiply by ten

K 7/6 7

Practical Points

Enough variables influence the tracing without introducing unnecessary technical ones Care should therefore be exercised to insure that technique is uniform from tracing to tracing and day to day so that allowances do not have to be made for variations in technique The following points are of importance

1 Effective *contact* between electrode and skin is essential Electrode jelly contains electrolytes and an abrasive the abrasive is intended to break down the waterproof horny layer of the skin so that the electrolytes of jelly and body may form a continuous conductor The jelly should therefore be rubbed briskly not delicately smeared on the skin before the electrode is applied Poor contact may lead to unduly low voltage and other artifacts in the tracing

2 *Standardization* should be consistent It should always if possible be full and should be adjusted exactly When 1 millivolt is thrown into the circuit the baseline should deflect exactly 10 mm If standardization varies from tracing to tracing it may be difficult to evaluate slight changes Moreover the interpreter is given considerable and unnecessary extra work if he has to take note of inconsistencies in standardization and make allowances for them

3 *Placement of the precordial electrode* is often too casual It should be as exact and constant as possible For this reason only bony landmarks should be used in locating the precordial points (page 2) Especially in leads close to the transitional zone (page 47 and figure 27) small displacements of the electrode may produce considerable changes in the pattern

4 *Position of patient* while the tracing is being taken is of importance He should be lying uniformly flat If for some reason he has to be in any other position a note to this effect should be made Lying on either side or sitting up usually alters the heart s axis electrical position and transitional zone thus serial tracings if taken in a variety of positions are difficult to compare

3

Complexes and Intervals

When a complex is partly above the baseline and partly below it it is called **diphasic or biphasic** When its excursions above and below the line are approximately equal it is called **isodiphasic or equiphasic**

P WAVE

This is the first wave of the electrocardiogram and represents the spread of the electrical impulse through the atria (**activation or depolarization** of atria) It is normally upright in leads 1 and 2 but is frequently diphasic or inverted in lead 3 It is normally inverted in aVR and upright in aVF It is variable in the other leads Its amplitude should not exceed 2 or 3 mm in any lead and its normal contour is gently rounded—not pointed or notched

Abnormalities that should be looked for are therefore

1 *Inversion* in leads where the P wave is normally upright or the presence of an upright P wave in aVR (where it should be inverted), such changes are usually found in conditions where the impulse travels through the atria by an unorthodox path—as in ectopic atrial or AV nodal rhythms (fig 11 C)
2 *Increased amplitude* which usually indicates atrial hypertrophy or dilatation and is found especially in mitral stenosis hypertension and cor pulmonale
3 *Increased width* usually indicates left atrial enlargement or diseased atrial muscle The normal P wave usually does not exceed 0 11 sec in duration
4 *Notching* when the left atrium is mainly involved (as in mitral

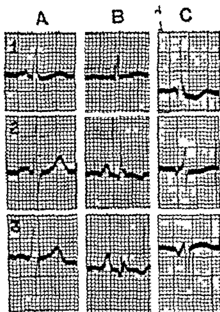


FIG 11 Abnormal P waves A **P mitrale** note broad notched P waves taller in lead 1 than in lead 3 B **P pulmonale** note flat P in lead with 1 with tall pointed P wave in leads 2 and 3 C **A V nodal rhythm** note inverted P in leads 2 and 3 with short P R interval

stenosis) the P wave often becomes wide and notched and is taller in lead 1 than in lead 3—P **mitrale** (fig 11 A) Notching is considered significant when the distance between peaks exceeds 0 04 sec

5 *Peaking* right atrial strain usually produces tall pointed P waves taller in lead 3 than in lead 1—P **pulmonale** (fig 11 B)

6 *Absence* of P waves which occurs in some A V nodal rhythms and in S A block

In summary P waves are

normally upright in 1 2 and aVF
normally inverted in aVR
variable in 3 aVL and chest leads

Abnormalities to be looked for are

1. abnormal inversion (or positivity in aVR)
2. increased amplitude and width
3. abnormal contour
4. absence

T_P WAVE

This wave, formerly called Ta (21), represents repolarization of the atria, and is in the direction opposite to the P wave—if the P wave is upright it is inverted, and vice versa (fig. 12 C). It is usually invisible because it coincides with the QRS complex. It can best be seen in complete A-V block, where the P waves are not followed by QRS complexes and there is consequently an opportunity for the T_P wave to show itself.

P-R INTERVAL

This is measured from the *beginning* of the P wave to the *beginning* of the QRS complex. It measures the time taken by the impulse to travel all the way from the S-A node to the ventricular muscle fibers, and this is normally from 0.12 to 0.20 seconds. It is customary to examine several intervals and record that which appears the longest. Other things being equal, the interval varies with heart size and rate, being shorter in smaller hearts or at faster rates. It is thus proportionately shorter in children, averaging 0.11 sec. at 1 year, 0.13 at 6 and 0.14 at 12 years. An interval prolonged beyond normal limits is regarded as evidence of A-V block (fig. 12 C).

At relatively slow rates a few apparently normal people with no evidence of heart disease have been found to have intervals ranging considerably above 0.20 sec. (23). Such prolongation is more likely to be a pointer to otherwise latent rheumatic or coronary disease, but one must not brand an individual as a "cardiac" whose only stigma is an unconventionally long P-R interval. Obviously it is a signal for the most thorough search to exclude cardiac abnormality, but if none other is found the heart should be acquitted with reservation. *At rates over 80* a P-R interval longer than 0.20 is probably always abnormal.

Biological values do not submit to arithmetical exactitude and it is important to bear in mind all the physiological factors which may influence P-R duration. An elephantine man with a correspondingly

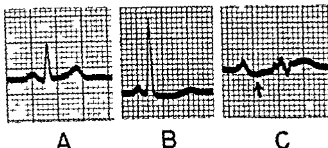


Fig 12 P R intervals A Normal interval of 0 14 sec B Short P R interval of 0 10 sec from a hypertensive patient shortly after an episode of atrial flutter C Prolonged P R interval of 0 30 sec note shallowly inverted Tp wave indicated by arrow immediately following P wave

large heart will have a longer interval than a petite woman of less than half his size She may have true block with an interval of only 0 19 while he may well have a normal duration of 0 21 sec Such biological variations too often are lost sight of in attempting to regiment natural values

The P R interval is abnormally short when the impulse originates in the A V node (fig 11 C) instead of the S A node and also when the passage of the impulse to the ventricle is accelerated as in the Wolff Parkinson White syndrome A short P R interval is also sometimes seen as a normal variation (fig 12 B) but this combination (normal P short P R and normal QRS) is perhaps not as benign a variant as might be thought because it is found often in association with hypertension (4) and with the tendency to develop paroxysms of tachycardia (3)

In summary the P R interval is

prolonged

(a) in A V block (p 131) due to coronary disease rheumatic disease, etc
(b) as a rare normal variation

shortened

(a) in A V nodal and low atrial rhythms (p 115-124)
(b) in Wolff Parkinson White syndrome (p 77)
(c) as a normal variation

The QRS COMPLEX represents the spread of the impulse through the ventricles and is the most important component of the tracing Its discussion will be postponed until later in this chapter (page 28ff)

ST SEGMENT

This segment is that part of the tracing immediately succeeding the QRS complex (fig 13) Two of its features should be observed 1) its *level* relative to the baseline i e whether it is elevated above or depressed below the T P segment and 2) its *shape*

Normally it is on the same level as the T P segment i e it is **iso electric** or only slightly above or below it It is sometimes normally elevated *not more than 1 mm in the standard leads and even 2 mm* in some of the chest leads it is never normally depressed more than half a millimeter or o An interesting exception is sometimes ob served particularly in healthy young Negroes (6 7) where the ST segments may be markedly elevated (sometimes as much as 4 mm) in one or more precordial leads (fig 14)

In shape the ST segment normally curves gently and imperceptibly into the proximal limb of the succeeding T wave It should not form a sharp angle with this limb nor should it pursue a frankly horizontal course Horizontality of the ST segment which is highly suspicious of coronary insufficiency has been called plane depression (fig 13 C)

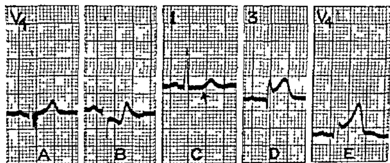


Fig 13 **ST segments** A Normal B Same lead same patient as A two minutes after exercise showing ST depression (coronary insufficiency) C ST segment is minimally depressed (certainly less than 0 5 mm) but is horizontal and forms a rather sharp angled junction with proximal limb of T wave (compare with A) D ST elevation from myocardial injury (acute infarction) E ST elevation as a normal variant in a healthy Negro

T WAVE

The T wave represents the recovery period of the ventricles when they recruit their spent electrical forces (**repolarization**) We particularly notice three of its features 1) its *direction*, 2) its *shape* and 3) its *height*

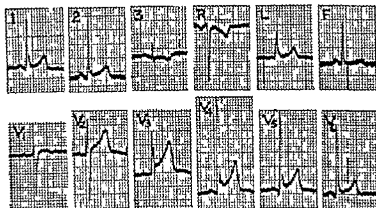


Fig 14 From a normal colored man of 29 years Note marked ST elevation in precordial leads especially V_2 and V_3

The T wave is normally upright in leads 1 and 2 and in chest leads over the left ventricle (except in infants and very young children) it is normally inverted in aVR, in all other leads it is variable Certain general rules govern this variability 1) The T wave is normally upright in aVL and in aVF if the QRS is more than 5 mm tall but may be inverted in the company of smaller R waves 2) In the precordial leads the tendency to inversion of T waves over the left ventricle (V_5 and V_6) rapidly diminishes with increasing age and in adult life it is generally considered abnormal if the T waves are inverted as far to the left as V_3 The T in V_1 may be inverted normally at any age and in V_2 it is also sometimes negative

It should be borne in mind that as mentioned earlier the polarity of the precordial waves tends to vary with the series of chest leads employed remembering the positive tendencies of the CR connection and the negative influence of CF it is obvious that an inverted T in CR_2 will be more surprising and more likely to be abnormal than an inverted T in CF_2 Indeed an inverted T in CF_2 is probably often within normal limits whereas with the V or even more the CR hookup an inverted T in this position is usually considered abnormal

We may summarize the direction of the normal adult T wave as follows

It is

normally upright in 1 2 and V_3–V_6
normally inverted in aVR
variable in 3 aVL aVF V_1 and V_2

The *shape* of the T waves is normally slightly rounded and slightly asymmetrical. When T waves are sharply pointed or grossly notched (8) they should be regarded with suspicion, though either of these characteristics may sometimes occur in precordial leads as a normal variant. Notching of the T waves is particularly common as a normal variant in children (fig 15); on the other hand it is not infrequently found in pericarditis. A sharply pointed symmetrical T wave (upright or inverted) is suspicious of myocardial infarction (fig 16).

The *height* of the T waves is also important. They are normally not above 5 mm in height in any standard lead and not above 10 mm in any precordial lead. Unusually tall T waves (fig 16 B and D) suggest myocardial infarction or potassium intoxication. Tall T waves are also not infrequently seen in myocardial ischemia without infarc-

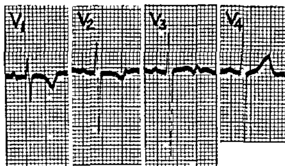


FIG 15 T waves in a normal child. Note marked notching in V_2; this is a normal transitional form of T wave between the normally inverted T in V_1 and the normally upright T in V_4.

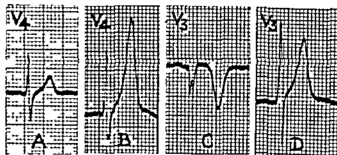


FIG 16 T waves. A Normal T wave. B Tall T wave of myocardial ischemia in a patient with angina but without infarction. C Deeply inverted symmetrical T wave of anterior infarction. D Tall upright symmetrical T

tion in certain forms of ventricular strain (see page 59) in psychotics and in patients with cerebro vascular accidents

Q T DURATION

This interval measured from the beginning of the QRS to the end of the T wave gives the total duration of ventricular systole It varies with heart rate sex and age and its normal values are most conveniently determined by consulting a prepared table based on the calculations of Ashman (table 2) A useful rule of thumb is that the Q T interval should be less than half the preceding R R interval This holds good for normal sinus rates However as the rate slows below 65 the maximal normal Q T duration fall further and further below half the preceding R P interval and as the rate increases above 90 the

TABLE 2

Normal Q T Intervals and the Upper Limits of the Normal

HEART RATE PER MIN	MEN AND CHILDREN	WOMEN	UPPER LIMITS OF THE NORMAL	
			Men and Children	Women
	sec			
40 0	0 449	0 461	0 491	0 503
43 0	0 438	0 450	0 479	0 491
46 0	0 426	0 438	0 466	0 478
48 0	0 420	0 432	0 460	0 471
50 0	0 414	0 425	0 453	0 464
52 0	0 407	0 418	0 445	0 456
54 5	0 400	0 411	0 438	0 449
57 0	0 393	0 404	0 430	0 441
60 0	0 386	0 396	0 422	0 432
63 0	0 3 8	0 388	0 413	0 423
66 5	0 370	0 380	0 404	0 414
70 5	0 361	0 3 1	0 395	0 405
75 0	0 352	0 362	0 384	0 394
80 0	0 342	0 352	0 374	0 384
86 0	0 332	0 341	0 363	0 372
92 5	0 321	0 330	0 351	0 360
100 0	0 310	0 318	0 338	0 347
109 0	0 297	0 305	0 325	0 333
120 0	0 283	0 291	0 310	0 317
133 0	0 268	0 276	0 294	0 01
150 0	0 252	0 258	0 275	0 282
172 0	0 234	0 240	0 255	0 262

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normal Q T duration gradually exceeds half the preceding R R These points will provide a near enough guide for most practical purposes The diagnostic value of the Q T duration is seriously limited by the technical difficulties of measuring it exactly (15)

The Q T duration is lengthened in congestive heart failure myocardial infarction (11) and hypocalcemia by quinidine and pronestyl It is sometimes lengthened in rheumatic fever (9 10 14) and in other causes of myocarditis (*12 13*) Careful measurement has shown that it is not lengthened in hypokalemia unless there is an associated deficit in calcium (16) It is shortened by digitalis and by potassium intoxication

U WAVE

This is a small wave of low voltage sometimes seen following the T wave Its normal polarity is the same as that of the T wave (i e when the T wave is upright it too is upright and vice versa) and the normal wave is often best discerned in lead V_3 It is rendered more prominent by potassium deficiency (fig 17) and its polarity is often reversed in myocardial ischemia and *left* ventricular strain (fig 17) These are the conditions in which its alterations are of most value in diagnosis but it is affected by numerous other factors digitalis quinidine, epinephrine, hypercalcemia thyrotoxicosis and exercise all increase its amplitude (17 19)

Its precise significance is uncertain In the cardiac cycle it coincides with the phase of supernormal excitability during ventricular recovery (18) and in this connection it is interesting to note that most ventricular premature beats occur at about the time of the U wave

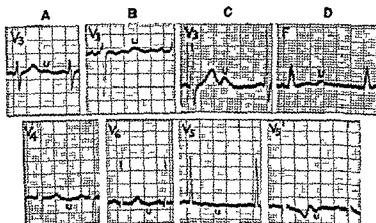


Fig. 17. U waves. Upper row—upright U waves. A, Normal. B, C and D, prominent U waves in hypokalemia. Lower row—inverted U waves. A, Tracing from which this is taken showed no abnormalities except for U wave inversion in several leads; this situation is referred to as "isolated U wave inversion." B, From a patient with hypertension whose tracing showed left ventricular strain including inverted U waves. C, From a patient with coronary insufficiency but without hypertension. D, Note marked inversion of T wave as well as U wave, from a hypertensive.

QRS COMPLEX

This complex is the most important in the electrocardiogram, as it represents spread of the impulse through the ventricular muscle (**activation** or **depolarization** of ventricles)

Proper labelling of the component waves of this complex must first be mastered (fig 18)

1 If the first deflection is downward (negative) it is a **Q wave**
2 The first upright deflection is an **R wave** regardless of whether or not it is preceded by a Q
3 A negative deflection following an R wave is called an **S wave**
4 Subsequent excursions above the line are labelled successively R′ R″ etc similarly later negative excursions are labelled S′ S″ and so on

If the QRS complex consists exclusively of an R wave the points at which the complex begins and ends are labelled Q and S respectively though there are no actual Q or S *waves* When the complex

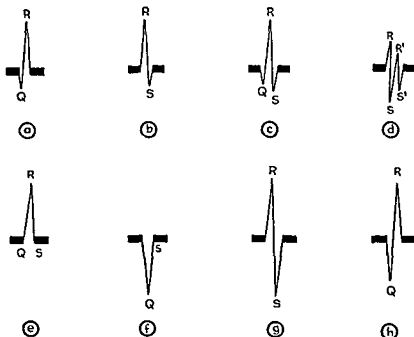


Fig 18 Labelled QRS complexes (see text)

consists exclusively of a Q wave it is usually described as a QS complex A convenient convention to save descriptive words is the use of small and large letters to signify the relative sizes of the component waves Thus in figure 18 (c) is conveniently labelled qRs which is quicker and simpler for the reader's eye than a small Q, a tall R and a small S wave In the figure (a) would be labelled qR (b) Rs (g) RS (h) QR, and so on The term QRS complex' may always be used as a sort of collective noun to describe the ventricular complex no matter what waves actually comprise it Thus all the examples in figure 18 may also quite correctly be labelled QRS

In interpreting the QRS complexes there are at least seven features that should be routinely inspected

1 their *duration* (the QRS interval)
2 their *voltage* or amplitude
3 the presence of *Q waves*
4 their *axis* in the standard limb leads
5 the *electrical heart position* (from the aV leads)
6 the relative prominence of the component waves in the precordial leads V_1 to V_6 noting the *transitional zone*
7 the timing of the *intrinsicoid deflections* in leads V_1 and V_6

Each of these will be discussed in turn

The *duration* of the normal QRS complex is usually given as 0 05 to 0 10 seconds but this is undoubtedly too restricted At times a short interval of not more than 0 04 is seen in normal hearts and occasionally an interval of 0 11 must be considered normal The QRS interval is measured from the beginning of the QRS to its end, and is usually estimated in the standard limb leads The chest leads frequently display a slightly longer QRS spread (0 01 or 0 02 seconds longer) than the standard leads the explanation for this is not clear A measurement of 0 12 seconds or more is indicative of abnormal intraventricular conduction and usually means block of one of the bundle branches or a ventricular arrhythmia

The *voltage* of the QRS complexes has wide normal limits It is generally agreed that if the total amplitude (above and below the isoelectric line) is 5 mm or less in all three standard leads it is too low to be healthy such low voltage is seen in diffuse coronary disease cardiac failure pericardial effusion myxedema primary amyloidosis and any other conditions producing widespread myocardial damage It is also found in emphysema generalized edema and obesity The minimal QRS amplitude in precordial leads waxes and wanes from

right to left across the chest being generally accepted as 5 mm in V_1 and V_6 7 mm in V_2 and V_5 and 9 mm in V_3 and V_4

It is more difficult to set an arbitrary upper limit to normal voltage Amplitudes up to 20 or even 30 mm are occasionally seen in lead 2 in normal hearts while the generally accepted maximum in a pre cordial lead is 25 to 30 mm

It must always be remembered that the amplitude or voltage ' re corded on the tracing is dependent on many factors besides the health of the heart for example the distance of the heart from the recording electrode (as determined by size of chest thickness of chest wall pres ence of emphysema etc) profoundly affects the size of the recorded deflections Such factors must always be given due consideration be fore the voltage of any complex is adjudged too high or too low

The significance of *Q waves* is one of the most important and some times the most difficult assessments in the tracing Size is important and yet a diminutive Q wave of less than a millimeter may be fraught with ominous significance while a QS complex of 10 mm in certain leads may sometimes be within normal limits A small Q wave of a millimeter or two is a normal finding in leads 1 aVL and aVF and in chest leads over the left ventricle e g V_6 The reason for this will become apparent in the discussion in Chapter 5 devoted to the relative prominence of the component waves in chest leads On the other hand deep QS or Qr complexes are a perfectly normal finding in aVR and QS complexes are occasionally found normally in lead 3 and in leads over the right ventricle V_1 and V_2 The Q wave should never take longer than 0 03 seconds to reach its lowest point (nadir) i e should not be more than 0 03 seconds in width To gauge their importance Q waves must be viewed in the light of the over all picture and one must take into account 1) their depth 2) their width 3) the leads in which they appear and most important 4) the clinical picture

REFERENCES

P WAVE

1 Thomas P and Dejong D The P wave in the electrocardiogram in the diagnosis of heart disease Brit Heart J 1954 16 241

P R INTERVAL

2 Alimurung M M and Massell B F The normal P R interval in infants and children Circulation 19 6 13 257

3 Lown B Ganong W B and Levine S A The syndrome of short P R

interval normal QRS complex and paroxysmal rapid heart action Circulation 195? 5 693
4 Scherf D Short P R interval and its occurrence in hypertension Bull N Y Coll Med 1941 4 116
5 Scherf D and Dix J H The effects of posture on A V conduction Am Heart J 195? 43 494

S T SEGMENT

6 Edeiken J Elevation of the RS T segment apparent or real in the right precordial leads as a probable normal variant Am Heart J 1954 48 331
7 Goldman M J RS T segment elevation in mid and left precordial leads as a normal variant Am Heart J 1953 46 817

T WAVES

8 Dressler W Roesler H and Lackner H The significance of notched upright T waves Brit Heart J 1951 13 496

Q T DURATION

9 Carmichael D B The corrected Q T duration in acute and convalescent rheumatic fever Am Heart J 1955 50 5 8
10 Craige E et al The Q T interval in rheumatic fever Circulation 1950 1 1338
11 Fick S R et al The Q T interval in myocardial infarction and left ventricular hypertrophy Am Heart J 1953 45 80
12 Fox T T et al The Q T interval in the electrocardiogram of children with tuberculo is Circulation 1950 1 1184
13 Gittleman I W Thorner M C and Griffith G C The Q T interval of the electrocardiogram in acute myocarditis in adults with autopsy correlation Am Heart J 1951 41 78
14 Kornel L and Braun K The Q T interval in rheumatic heart disease Brit Heart J 1956 18 8
15 Lepeschkin E and Surawicz B The measurement of the Q T duration of the electrocardiogram Circulation 195? 6 378
16 Lepeschkin E and Surawicz B The duration of the Q U interval and its components in electrocardiograms of normal persons Am Heart J 1953 46 9

U WAVE

17 Lepeschkin E The U wave of the electrocardiogram Arch Int Med 1955 96 600
18 Mack I Langendorf R and Katz L N The supernormal phase of recovery of conduction in the human heart Am Heart J 1947 34 374
19 Palmer J H Isolated U wave negativity Circulation 1953 7 205

QRS INTERVAL

20 Lepeschkin E and Surawicz B The measurement of the duration of the QRS interval Am Heart J 195? 44 80

GENERAL

21 Committee on Electrocardiography American Heart Association Recommendations for standardization of electrocardiographic and vectorcardiographic leads Circulation 1954 10 564
22 Kossman C E The normal electrocardiogram Circulation 1953 8 920
23 Manning G W Electrocardiography in the selection of Royal Canadian Air Force aircrew Circulation 1954 10 401
24 Packard J M Graettinger J S and Graybiel A Analysis of the electrocardiograms obtained from 1000 young healthy aviators Ten year follow up Circulation 1954 10 384

Some Electrocardiographic Milestones

1858 Kolliker and Muller in Germany demonstrated that contraction of heart muscle was accompanied by electrical activity

1887 Waller in England recorded the first electrocardiogram in man using a capillary electrometer

1903 Einthoven in Holland introduced the string galvanometer electrocardiograph and employed the classical standard limb leads in human electrocardiography

1914 Lewis in England introduced a two string electrocardiograph Our knowledge of the sequence of myocardial activation is based on his studies with this machine Lewis also introduced the concept of the intrinsic deflection

1932 Wolferth and Wood in America demonstrated the value of precordial leads

1934 Wilson in America introduced the central terminal and with it the unipolar or V leads

1935 Wilson in America demonstrated the superiority of multiple precordial leads over one such lead

4

Axis Deviation and Electrical Position

AXIS DEVIATION OF QRS COMPLEXES

If the main QRS deflections in leads 1 and 3 are directed upwards (i e are positive) it is termed a **normal axis** If on the other hand the main QRS deflection in lead 1 is upward (positive) while the main deflection in lead 3 is downward (negative) the pattern is described as **left axis deviation** If the main deflection in lead 1 is negative while that in lead 3 is positive we call it **right axis deviation** It is also convenient though not strictly accurate to regard it as a minor degree of right axis deviation when the QRS in both 1 and 3 are up right but that in lead 3 is taller than that in lead 1—i e if the QRS complex in lead 1 is less positive than in lead 3 These patterns are illustrated in figure 19

It should be emphasized at once that deviation of the axis to the left or right is not necessarily abnormal normal axis is not the only norm There are generally accepted upper limits of normal de viation to both right and left and certainly deviation beyond these extremes may be considered as abnormal Thus the axis of the heart or the degree of axis deviation can be calculated from the following formula

$$(R_1 + S_3) - (S_1 + R_3) = \text{axis}$$

The upright wave of lead 1 measured in millimeters is added to the downward deflection of lead 3 from this sum is subtracted the sum of the downward deflection in lead 1 and the upward deflection in lead 3 In other words the tendency to right axis is subtracted from the tend ency to left axis deviation If the axis comes to +30 or more it is con sidered abnormal left axis deviation if it comes to −10 or less it is considered abnormal right axis shift

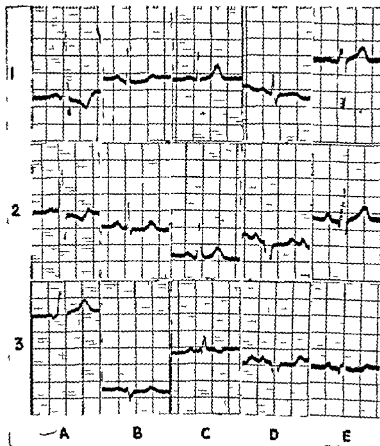


FIG 19 Axis of the QRS A Marked left axis deviation B Slight left axis deviation C Normal axis D Slight right axis deviation E Marked right axis deviation

Many deviations short of these extremes however are at times abnormal If for example a person who has had a normal axis all his life develops hypertension and his axis begins to shift to the left the earliest indication of this axis shift may be presumed to result from his hypertension and therefore is an expression of abnormality in the individual Another person may have shown a normal degree of left axis deviation all his life He then develops emphysema and the axis begins to shift away from the left towards the right On the way to frank right axis deviation it will pass through the stage we

call normal axis yet this normal axis being caused by the development of hypertrophy of his right ventricle as a result of the emphysema is actually abnormal for this man who has always had a left axis shift

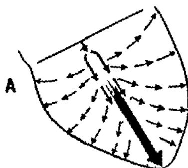
Bearing such reservations in mind we may summarize the circumstances in which axis shift is found

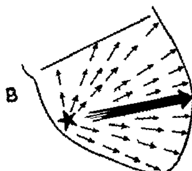
Right	*Left*
Normal variation	Normal variation
Mechanical shifts—inspiration emphysema	Mechanical shifts—expiration high diaphragm from pregnancy ascites abdominal tumors etc
Right ventricular hypertrophy	Left ventricular hypertrophy
Right bundle branch block	Left bundle branch block
Left ventricular premature beats	Right ventricular premature beats
Inferior infarction	Anterolateral infarction
Dextrocardia	

In considering mechanical shifts it should be noted that such disturbances as pneumothorax and pleural effusion usually cause a wholesale shift of the mediastinum heart and all towards the opposite side and do not necessarily affect the heart's axis—they push it to one side without necessarily rotating it

The reason why ectopic ventricular beats and bundle branch blocks swing the electrical axis of the heart in the direction they do is easily explained The normal impulse spreads down between the two ventricles and then fans out simultaneously to both sides activating the ventricles in parallel The resultant direction of spread is therefore approximately in the axis between the ventricles (fig 20 A) If on the other hand the impulse begins in one ventricle and spreads to the other thus activating the ventricles in series the resultant direction of spread is obviously swung towards the second ventricle If there is an ectopic focus in the *right* ventricle giving rise to premature beats the impulse spreads predominantly from right to left swinging the axis for that beat to the left (fig 20 B) If the *left* bundle branch is blocked a rather similar state of affairs exists for again the impulse spreads through the right ventricle first and then involves the left (fig 20 C)

It has been said that determination of the electrical axis of the heart is of little if any value in diagnosis This is not true because at times it affords a most important diagnostic clue Two examples of its value will illustrate this 1) in a congenital heart with cyanosis the presence of left axis deviation makes the diagnosis of tricuspid atresia





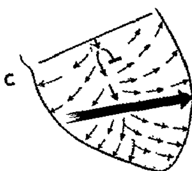


FIG. 20 Left axis shift in right ventricular premature beat and left bundle branch block

2) the presence of left axis deviation may be an important constituent of the pattern of anterolateral infarction (page 166)

AXIS DEVIATION OF P WAVES

Now that axis deviation of the QRS is appreciated it can be seen that the pattern of right atrial hypertrophy already referred to (page 19) with P_1 lower than P_3 is an expression of a tendency towards right axis deviation of the atria and similarly the P wave pattern characteristic of mitral stenosis with P_1 taller than P_3 and P_3 sometimes actually inverted indicates a shift of the atrial axis towards the left

ELECTRICAL HEART POSITION

The five generally recognized positions about the heart's antero posterior axis are **horizontal semi-horizontal intermediate, semi vertical** and **vertical** These positions are recognized entirely from aVL and aVF The five positions are illustrated in figure 21 If the main deflection of the QRS is positive in both leads the position is called intermediate If the main deflections are divergent the heart is horizontal, if convergent vertical Semi horizontal and semi vertical positions are halfway stations between the intermediate position and the horizontal and vertical extremes

It is helpful to correlate the patterns of axis deviation with the patterns of position It is simple enough to associate left axis deviation with a horizontal heart and right axis deviation with a vertical heart If leads 1 and 3 in axis deviation are compared with leads aVL and aVF in horizontal and vertical hearts it will be immediately obvious that the QRS complexes are divergent in both left axis and horizontal patterns while they are convergent in both right axis and vertical patterns (fig 22)

NORMAL FINDINGS IN aV LEADS

aVR All three complexes—P QRS and T—are inverted This is to be expected for as aVR is derived from the right shoulder and as the general direction of spread of both atrial and ventricular impulse is away from the right shoulder obliquely downwards towards the left leg one would expect both P and QRS deflections to be negative The inverted QRS complex usually presents an rS pattern but may be QS or QR in form

aVL All the complexes in this lead are variable P QRS and T all may be upright or inverted according to the heart's electrical position If the QRS is as much as 6 mm tall the accompanying T wave should not be inverted Any pattern of the QRS can be normal even QS when the voltage is low However, if the R wave is 6 mm or more the Q wave should be small by comparison—not more than 1–2 mm deep and not more than 0 03 seconds in duration

aVF The complexes in this lead are also variable depending mainly on the heart's position The P wave is usually upright but may at times be inverted Again as in aVL

ELECTRICAL HEART POSITION

The five generally recognized positions about the heart's antero-posterior axis are **horizontal**, **semi horizontal**, **intermediate**, **semi vertical** and **vertical**. These positions are recognized entirely from aVL and aVF. The five positions are illustrated in figure 21. If the main deflection of the QRS is positive in both leads the position is called intermediate. If the main deflections are divergent the heart is horizontal; if convergent, vertical. Semi horizontal and semi vertical positions are halfway stations between the intermediate position and the horizontal and vertical extremes.

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NORMAL FINDINGS IN aV LEADS

aVR All three complexes—P, QRS and T—are inverted. This is to be expected, for as aVR is derived from the right shoulder and as the general direction of spread of both atrial and ventricular impulse is away from the right shoulder obliquely downwards towards the left leg, one would expect both P and QRS deflections to be negative. The inverted QRS complex usually presents an rS pattern, but may be QS or QR in form.

aVL All the complexes in this lead are variable. P, QRS and T all may be upright or inverted, according to the heart's electrical position. If the QRS is as much as 6 mm. tall the accompanying T wave should not be inverted. Any pattern of the QRS can be normal, even QS when the voltage is low. However, if the R wave is 6 mm. or more the Q wave should be small by comparison—not more than 1–2 mm. deep and not more than 0.03 seconds in duration.

aVF The complexes in this lead are also variable depending mainly on the heart's position. The P wave is usually upright but may at times be inverted. Again, as in aVL

ELECTRICAL HEART POSITION

The five generally recognized positions about the heart's antero posterior axis are **horizontal semi horizontal, intermediate semi vertical** and **vertical** These positions are recognized entirely from aVL and aVF The five positions are illustrated in figure 21 If the main deflection of the QRS is positive in both leads the position is called intermediate If the main deflections are divergent the heart is horizontal if convergent vertical Semi horizontal and semi vertical positions are halfway stations between the intermediate position and the horizontal and vertical extremes

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aVF The complexes in this lead are also variable depending mainly on the heart's position The P wave is usually upright but may at times be inverted Again as in aVI

5

Genesis of the Precordial Pattern The Intrinsicoid Deflection

Over the normal heart the R wave becomes taller and the S wave smaller as the electrode is moved from right to left across the chest To understand this it is helpful to consider the patterns that result when an electrode is placed at various points along a single strip of stimulated muscle

In figure 23 the muscle strip ABC is stimulated at the arrow and the wave of activation spreads from left to right to the other end of the strip If the electrode is placed successively at points 1 2 and 3 the illustrated patterns are respectively derived from point 1 an rS complex from point 2 an RS and from point 3 an Rs complex It is easy to deduce from these patterns that as long as the impulse is travelling towards the electrode a positive deflection (R wave) is produced while a negative deflection (S wave) is inscribed when the impulse has passed the electrode and is travelling away from it

A convenient way to rationalize this finding is to think of the impulse as a moving dipole i e a pair of charges one positive and one negative travelling together with the positive charge always leading This is a crude but convenient approximation of what actually occurs when an impulse travels through stimulated tissue Let us consider in terms of the dipole what happens when the electrode is placed in the middle of the muscle strip (point 2) and the strip is then stimulated As the dipole travels from left to right the leading positive charge gets nearer and nearer to the recording electrode as it approaches it exerts a stronger and stronger influence on the electrode

5

Genesis of the Precordial Pattern The Intrinsicoid Deflection

Over the normal heart the R wave becomes taller and the S wave smaller as the electrode is moved from right to left across the chest To understand this it is helpful to consider the patterns that result when an electrode is placed at various points along a single strip of stimulated muscle

In figure 23 the muscle strip ABC is stimulated at the arrow and the wave of activation spreads from left to right to the other end of the strip If the electrode is placed successively at points 1, 2 and 3 the illustrated patterns are respectively derived from point 1 an rS complex from point 2 an RS and from point 3 an Rs complex It is easy to deduce from these patterns that as long as the impulse is travelling towards the electrode a positive deflection (R wave) is produced while a negative deflection (S wave) is inscribed when the impulse has passed the electrode and is travelling away from it

A convenient way to rationalize this finding is to think of the impulse as a moving dipole i e a pair of charges one positive and one negative travelling together with the positive charge always leading This is a crude but convenient approximation of what actually occurs when an impulse travels through stimulated tissue Let us consider in terms of the dipole what happens when the electrode is placed in the middle of the muscle strip (point 2) and the strip is then stimulated As the dipole travels from left to right the leading positive charge gets nearer and nearer to the recording electrode as it approaches it exerts a stronger and stronger influence on the electrode

5

Genesis of the Precordial Pattern The Intrinsicoid Deflection

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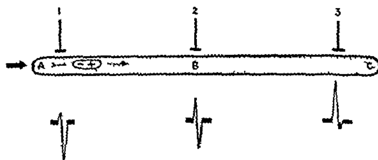


Fig 23

and the tracing becomes more and more positive until it reaches maximal positivity (peak of R wave) at the moment that the positive charge is immediately under the electrode A split second later the dipole has moved on and the negative charge is now immediately *under the electrode exerting its maximal influence* So the tracing makes a quick swing (the downstroke) from maximal positivity to maximal negativity Then as the dipole continues on its journey its negative tail recedes from the electrode and its influence thereby diminishes The tracing becomes less and less negative until when the whole muscle strip has been activated the tracing regains the isoelectric line

The downstroke which represents the abrupt swing from maximal positivity to maximal negativity is called the **intrinsic deflection** It is a deflection of great practical importance for it tells us the moment that the impulse (dipole) has arrived under the electrode We know that the start of the upstroke marks the moment that the impulse started from the arrow we now also know that the start of the downstroke mark the moment that the impulse arrived at B We can thus measure the time it takes for the impulse to travel the distance from the arrow to B This of course like all timing is measured horizontally as illustrated in figure 24 This simple principle has been used for decades in experimental physiology Lewis used it to plot the times of impulse arrival at various points in the atria when he was attempting to prove his theory of circus movement Prinzmetal has recently used it even more extensively in his experiments on the atrial arrhythmias

In the light of the foregoing let us now examine what happens when electrodes are placed in contact with the myocardium of normally

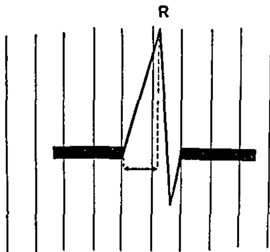


FIG 24 Timing of the intrinsic deflection The time that elapses from the beginning of the QRS complex to the peak of the R wave is measured horizontally

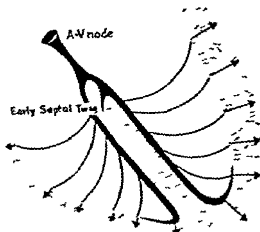


Fig 25 Schematic drawing of ventricular conduction paths Note 1) the early septal twig arising from the left bundle branch high up and 2) perpendicular path of impulse through myocardium

functioning ventricles either in the experimental animal or in man with the heart surgically exposed If an electrode is placed in contact with the surface of the right ventricle a mainly negative (rS) deflection is inscribed if placed in contact with the left ventricle a mainly positive (qR) complex is registered For all practical purposes the patterns are the same as those obtained clinically from precordial points to the right and left say V_1 and V_6 To appreciate the reason for this it is necessary to recall the ramifications of the ventricles conduction paths These are schematically represented in figure 25

Most of the details of the spread of the cardiac impulse are well enough known but two points deserve emphasis 1) Shortly after the bundle of His has divided into its branches the left bundle branch gives off a small twig to the upper part of the septum the **early septal twig** No corresponding twig arises from the right bundle branch It follows that the first part of the ventricular myocardium to be activated is the upper part of the septum and this is activated exclusively from left to right 2) When the impulses are distributed by the Purkinje fibers to the endocardial surfaces of the ventricular wall they then travel perpendicularly through the walls from endocardium to epicardium

We are now in a position to see why right and left precordial leads develop their respective patterns Figure 26 illustrates diagrammatically the order in which various areas of ventricular muscle are activated First the upper part of the septum exclusively from the left (1) then the lower part of the septum simultaneously from both sides (2) then both ventricular walls are activated simultaneously but because the right wall is much thinner than the left the impulse traverses its perpendicular path through the right wall and arrives at the epicardium (3) long before impulses on the left have reached the epicardial surface Then finally the left ventricular muscle is penetrated first at the apex and then successively towards the base (4 5 6)

Now if an electrode is placed over the thin wall of the right ventricle (A in fig 26), the first impulse (dipole) to influence it will be that derived from the early septal twig (1) this is travelling towards the electrode and the deflection produced will therefore be positive The impulses at 2 are travelling in opposite directions and are equidistant

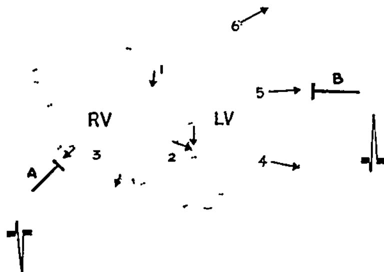


Fig 26 The approximate order of activation of the ventricular myocardium and the form of ventricular complex derived over each ventricle

from A so that they neutralize each other and exert no discernible influence on the tracing. The next impulse (3) is also travelling towards A and therefore augments the already positive deflection. From this time on the only dipoles left in the picture are those activating the left ventricle. These are all travelling away from the electrode A and therefore cause a negative deflection. The wall is thick and the impulses have a relatively long journey so the S wave is relatively deep. The composite picture produced is thus an rS complex.

When an electrode is placed over the left ventricle (B in fig. 26) again the first influence felt is (1). This is now travelling away from our electrode and therefore causes a small initial negative deflection (Q wave). The impulses at (2) cancel each other out as before. From now on the electrode is under the influence of the approaching impulses travelling towards it through the left ventricular wall. Therefore the remainder of the left ventricular tracing is positive and the composite picture is a qR complex.

Thus over the right ventricle a deep S wave represents activation of the left ventricle while over the left ventricle itself its activation is represented by a tall R wave. In other words from both sides of the heart the major deflection represents activation of the major (left) ventricle. As in the single muscle strip the downstroke from the peak of the R wave is the intrinsic deflection and tells us when the impulse has reached the epicardial surface of the ventricle over which the electrode is placed. As the right ventricle has a much thinner wall the impulse over this ventricle naturally reaches the surface much earlier than it reaches the surface of the left ventricle, i.e. the peak of the small R wave over the right ventricle is reached earlier than the peak of the tall R wave over the left ventricle.

In clinical practice we obviously cannot take **direct or epicardial leads** and the best we can do is to place the electrode on the chest wall at strategic intervals across the precordium. The resulting series of **semi direct or precordial leads** produces patterns very similar to those taken with the electrode in direct epicardial contact. In these clinical leads the downstroke is the analogue of the intrinsic deflection and is therefore called the **intrinsicoid deflection**. This deflection should begin, i.e. the peak of the R wave should be reached, within 0.02 seconds in V_1 and within 0.04 seconds in V_6. If it takes longer than this for the intrinsicoid deflection to start downwards it means that the impulse is late in reaching the epicardial surface of the ventricle under the electrode and this indicates either that the wall of the

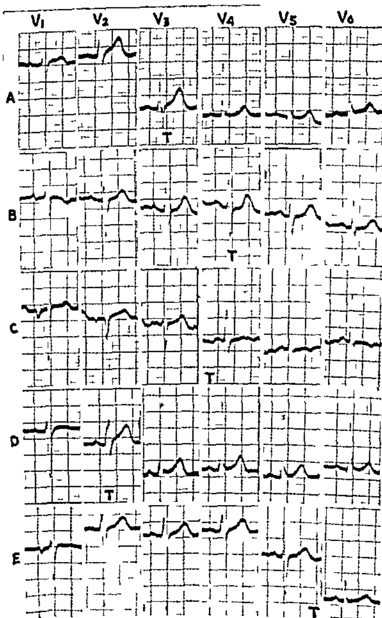


Fig [illegible] Transitional zones (T) A B C Normal transitions D Counter clockwise rotation E Clockwise rotation

ventricle has become thickened (ventricular hypertrophy) or dilated (so that the conducting paths have been lengthened) or that there is a block in the conducting system to the ventricle concerned (bundle branch block) The intrinsicoid deflection is therefore a most useful component of the ventricular tracing

This application of the dipole concept to the complex process of activation of the entire heart is obviously an over simplification though a most convenient and practical one Regardless of what term —impulse dipole electromotive force or vector—is used in describing the phenomena of myocardial activation the principles enunciated above are most helpful in visualizing the train of electrical events

CLOCKWISE AND COUNTER CLOCKWISE ROTATION

Between the definite right ventricular pattern (rS) of V_1 and the definite left ventricular pattern (qR) of V_6 there are transitional patterns—as was stated above the S wave becomes less deep as the electrode is moved towards the left while the R wave becomes progressively taller The **transitional zone** is the area in which the QRS complex is equiphasic (an RS complex) and this usually appears in V_3 or V_4 or between them In figure 27 five series of precordial leads from V_1 to V_6 are recorded The first three show normal transitional zones (T) in A lead V_3 shows the equiphasic complex in B lead V_4 shows it in C the actual transitional pattern is not shown but V_3 looks like a right ventricular lead while V_4 looks like a left ventricular lead the transition from one to the other has occurred between the two

The last two series in the figure show abnormal transitions In D it can be seen that the transition occurs between V_1 and V_2 in other words the left ventricular pattern is recorded unusually far to the right of the chest In E the transition occurs between V_5 and V_6 i e a right ventricular pattern is recorded unusually far to the left of the precordium

In explaining this we picture the heart to have rotated about its longitudinal axis In describing rotation about this axis we are asked to look *up* at the heart from *under* the diaphragm Thus if the front of the heart revolves towards the left we have from our subphrenic viewpoint **clockwise rotation** If the front of the heart rotates towards the right we have **counter clockwise rotation** Clockwise rotation will obviously move the zone between the two ventricles towards the left so that the transitional zone in the precordial tracing

shifts to the left (E in fig 27) while counter clockwise rotation will shift the transitional zone towards the right (D in fig 27) Such rotations are not necessarily abnormal

Rarely in extreme cases the transitional zone between the two ventricular patterns may be situated further to the right than V_1 or further to the left than V_6 In such situations leads further to the right (V_{3R} and V_{4R}) or further to the left (V_7 and V_8) may be taken in an attempt to obtain right or left ventricular patterns respectively

NOTE ON USE OF TERMS VENTRICULAR LEAD AND VENTRICULAR PATTERN

It should be emphasized however that a precordial record is not derived exclusively from one underlying area of the myocardium No matter what lead is used the resulting tracing is always a composite picture an electrical resultant of all the many simultaneous impulses or dipoles that are travelling in various directions through the whole myocardium It is true that the area of myocardium subjacent to the electrode may make a major contribution to the record—the so called local pick up effect—because an important factor determining recorded voltage is the nearness of the electrode to the electrical force and therefore a nearby force will exert more influence than a distant one on the pattern produced As an example if the electrode is placed at position 6 the impulses travelling through the wall of the left ventricle are appreciably nearer to it than those traversing the right ventricular wall and the left ventricle therefore exerts a greater influence than the right but as long as forces continue to be generated in the right ventricular wall they will be making some contribution to what we have been calling the left ventricular pattern In other words a right or left ventricular pattern means the type of pattern produced *when the electrode is placed over* the right or left ventricle rather than the pattern produced *exclusively by* the subjacent ventricle Indeed it is obvious that the so called right ventricular pattern is mainly derived from left ventricular forces Provided it is fully appreciated what is meant by right and left ventricular lead and right and left ventricular pattern these are useful descriptive terms

REFERENCES

1 Grant R P The relationship between the anatomic position of the heart and the electrocardiogram A criticism of unipolar electrocardiography Circulation 1953 7 890

2 Milnor W R Talbot S A and Newman E V A study of the relationship between unipolar leads and spatial vectorcardiograms using the panoramic vectorcardiograph Circulation 1953 7 545

6

Ventricular Hypertrophy and Strain

With the genesis of the normal precordial tracing fresh in mind the readiest pattern to turn our attention to is that of ventricular hypertrophy.

LEFT VENTRICULAR HYPERTROPHY AND STRAIN

The pattern of left ventricular hypertrophy is exactly what one would predict. If the wall of the left ventricle is thicker than normal the impulse will take longer to traverse it and arrive at the epicardial surface. Therefore the QRS interval will increase towards or to the upper limit of normal; the intrinsicoid deflection may be somewhat delayed over the left ventricle and the voltage of the QRS complexes will increase—producing deeper S waves over the right ventricle and taller R waves over the left (as in the precordial leads in fig. 28). The most reliable single indication of left ventricular hypertrophy is present when the sum of the S wave in V_1 and the R wave in V_5 totals more than 30 mm. In figure 28 the total is 48 mm. Accompanying these precordial changes, left axis deviation in the standard leads is usually found.

When strain complicates the picture, the ST segments and T waves are affected. Over the left ventricle (V_4, V_5, V_6) the ST segments become depressed with an *upward convexity* whose final downward curve blends into an inverted T wave. If left axis deviation is present, the same ST-T changes are usually evident in lead I; similar changes also appear in aVL leads having the form (qR) of left ventricular leads. In the case illustrated in figure 28, these changes have taken place in aVL. Not uncommonly the earliest sign of left ventricular strain is inversion of T waves in left ventricular leads.

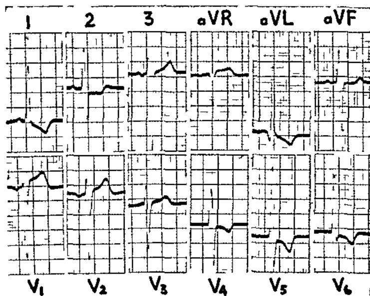


Fig 28 **Left ventricular hypertrophy and strain** Note horizontal heart with left axis deviation ST T changes in 1 ₋ aVL V₄₆ high voltage of QRS complexes in precordial leads

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It is important to realize that left axis deviation is not an invariable accompaniment of left ventricular hypertrophy It is in fact possible to have actual right axis deviation in such a heart if it is in a vertical position (fig 29) If the ST T changes of strain are present they will then be seen in whichever standard leads have the form of left ventricular leads Thus when left ventricular hypertrophy and strain appear in a horizontal heart with left axis deviation—the usual finding —lead 1 (and perhaps 2) and aVL will show ST T changes (fig 28) whereas in a vertical heart with right axis deviation the ST T changes will appear in lead 3 and aVF (fig 29) If the tracing shows tall R waves in all three standard leads as in normal axis or slight right axis deviation (fig 29) the ST T changes may be present in all three standard leads Further examples of left ventricular hypertrophy and strain are shown in figures 30 and 31

In summary heart position alone determines *which* standard and aV leads will manifest the ST T changes of strain and the diagnosis of left ventricular strain must be made predominantly from the chest leads



Fig 29 **Left ventricular hypertrophy and Strain** Note *vertical* heart with *right* axis deviation therefore ST T changes in 2 3 aVF V6 Notice QRS amplitude of over 40 mm in V and V

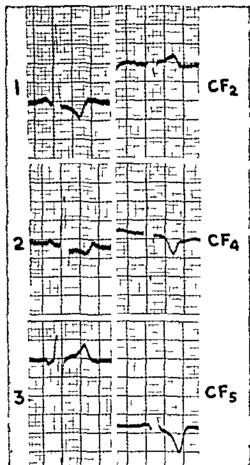


Fig 30 **Left ventricular hypertrophy and strain** Note marked left axis deviation with ST T changes in 1 2 CF_4 and CF delayed intrinsicoid deflection in CF_5

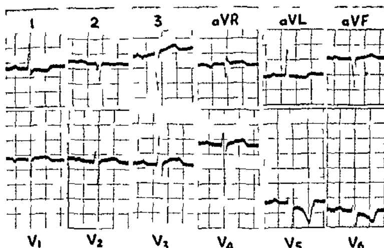


Fig 31 **Left ventricular hypertrophy and strain** Note left axis deviation ST T changes in I aVL V_4 and V

Strain is a useful non committal term The exact mechanism which produces its pattern is not completely settled but there are several factors believed to contribute to it It is known to develop in those who have shown the pattern of left ventricular hypertrophy for some time and the pattern intensifies when dilatation and failure set in Myocardial ischemia and slowing of intraventricular conduction are important among the factors which probably contribute to the pattern

Along with the left ventricle the left atrium may also feel the strain In such cases the P mitrale pattern (page 19) with left axis deviation of the P waves may be associated with the pattern of ventricular hypertrophy and strain

Much the commonest cause of left ventricular hypertrophy and strain is hypertension Less frequently the pattern is found following myocardial infarction in aortic stenosis aortic insufficiency coarctation of the aorta and occasionally in other conditions

Salient features of left ventricular hypertrophy and strain

1 Deeper S waves over right ventricle and taller R waves over left
2 QRS interval towards upper limit of normal (0·08–0·11 sec)
3 Late intrinsicoid deflection in $V_{5,6}$
4 Left axis deviation—usually
5 ST segment depression with *upward convexity* in left precordial leads and in whichever standard and aV leads show left ventricular (qR) pattern

RIGHT VENTRICULAR HYPERTROPHY AND STRAIN

The pattern of right ventricular hypertrophy and strain is not so easily rationalized but it is easily enough remembered In the fully developed picture the precordial pattern is reversed so that tall R waves appear over the right precordium (V_1 V_2), while rS complexes develop over the left ventricle The simplest and possibly the correct way to rationalize this reversal is to think of precordial complexes with tall R waves as representing the *major* rather than the left ventricle Normally the left ventricle is the major ventricle and so produces tall R waves When the right ventricle shows extreme hypertrophy it becomes the major ventricle and the rS pattern appears over the now minor left ventricle The QRS complex may become somewhat prolonged and the intrinsicoid deflection somewhat delayed over the right side of the precordium but these changes are not usually so marked as the corresponding changes in hypertrophy of the left ventricle In the standard leads right axis deviation almost invariably occurs

Right ventricular strain manifests itself in ST T changes similar to those seen in left ventricular strain but in different leads namely in those over the right ventricle (V_1 V_2) and in lead 3 The changes of fully developed right ventricular hypertrophy and strain are seen in figure 32

This full blown pattern of right ventricular hypertrophy and strain is much less commonly seen than that of left ventricular strain because the causes of right strain are less common and because it requires a higher degree of strain to produce the mature pattern In left ventricular hypertrophy the left ventricle is already the major' ventricle and as it hypertrophies its majority becomes accentuated so that early hypertrophy is fairly readily seen as an exaggeration of the normal pattern In right hypertrophy on the other hand the right ventricle starting as the minor ventricle has a good deal of overtaking to do before it becomes the major ventricle and materially alters the tracing

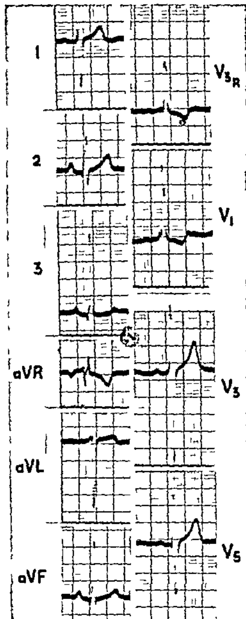


Fig 3? **Right ventricular hypertrophy and strain** Note marked right axis deviation tall R waves over right ventricle with deep S wave over left ST T changes only slight in 3 but well developed in V_{3R} and V

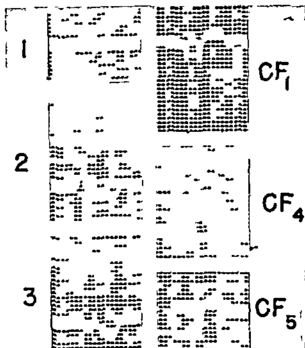


FIG 33 Tracing from patient with emphysema Note right axis deviation P pulmonale pattern ST T changes in 2 and 3 and deep S waves as far to the left as CF (clockwise rotation)

A pattern short of the fully developed right ventricular strain pattern is more often seen This consists in rS complexes all across the precordium (clockwise rotation) with right axis deviation in the standard leads Such a tracing is seen in many cases of emphysema for example which have not yet developed severe cor pulmonale A tracing from such a patient is seen in figure 33

In the presence of right ventricular hypertrophy and strain the right atrium may al o feel the strain In such cases the P pulmonale pattern is added to the pattern of ventricular strain If pure mitral stenosis is the cause of the right ventricular strain the P mitrale pattern may appear

The main causes of right ventricular hypertrophy and strain are congenital lesions such as the tetralogy of Fallot pulmonary stenosis and transposition of the great vessels acquired valvular lesions including pure mitral stenosis tricuspid stenosis and/or insufficiency and chronic lung diseases especially emphysema

PATTERNS OF SYSTOLIC AND DIASTOLIC OVERLOADING

Recently the patterns of ventricular strain have been subdivided into two types systolic overloading and diastolic overloading (1) When the heart has to pump against an obstruction it is in systole that the strain is felt when the blood overfills the ventricle as in aortic insufficiency the predominant strain is obviously felt in diastole

With systolic overloading of the left ventricle as seen in hypertension and aortic stenosis the classical pattern of hypertrophy and strain as outlined on page 50 is seen but when the main load is borne in diastole as in pure rheumatic aortic insufficiency or in patent ductus a different pattern is seen this consists of prominent upright T waves as well as tall R waves over the left ventricle (V_{5-6})

With systolic overloading of the right ventricle as seen in pulmonic stenosis or pulmonary hypertension the classical pattern seen in figure 32 is produced but when the main load is diastolic as in atrial septal defect the pattern of complete or incomplete right bundle branch block (see next chapter) results This pattern apparently does not result from blockade of the right bundle branch but rather results from hypertrophy of the basal portions of the right ventricle (8)

Salient features of right ventricular hypertrophy and strain

1 Reversal of precordial pattern with tall R over right precordium (V_1 V_2) and deep S over left (V_5 V_6)
2 QRS interval within normal limits
3 Late intrinsicoid deflection in $V_{1\ 2}$
4 Right axis deviation
5 ST segment depression with *upward convexity* and inverted T waves in right precordial leads ($V_{1\ 2}$) and in whichever standard and aV leads show tall R waves

REFERENCES

1. Cabrera E and Monroy J R Systolic and diastolic loading of the heart II Electrocardiographic data Am Heart J 1952 43 669
2. Johnson J B et al The relation between electrocardiographic evidence of right ventricular hypertrophy and pulmonary artery pressure in patients with chronic pulmonary disease Circulation 1950 1 536
3. McGregor M The genesis of the electrocardiogram of right ventricular hypertrophy Brit Heart J 1950 12 351
4. Myers G B The form of the QRS complex in the normal precordial electrocardiogram and in ventricular hypertrophy Am Heart J 1950 39 637
5. Scott R C et al Left ventricular hypertrophy A study of the accuracy of current electrocardiographic criteria when compared with autopsy findings in one hundred cases Circulation 1955 11 89
6. Walker I C Helm R A and Scott R C Right ventricular hypertrophy I Correlation of isolated right ventricular hypertrophy at autopsy with the electrocardiographic findings Circulation 1955 11 215
7. Walker I C Scott R C and Helm R A Right ventricular hypertrophy II Correlation of electrocardiographic right ventricular hypertrophy with the anatomic findings Circulation 1955 11 223
8. Walker W J et al Electrocardiographic and hemodynamic correlation in atrial septal defect Am Heart J 1956 52 547

7

Intraventricular Block

It is convenient to consider intraventricular block next because its patterns in many ways are simply exaggerations of the corresponding ventricular hypertrophy and strain pattern The commonest form of intraventricular block is bundle branch block but it may also result from diffuse slowing of the impulse throughout the conduction system of one ventricle or by conduction disturbances in the ventricular wall

LEFT AND RIGHT BUNDLE BRANCH BLOCKS

If one of the branches of the bundle of His is blocked by disease it stands to reason that the impulse will travel down the branch to the other ventricle first Having activated this ventricle the impulse will spread through the septum to the ventricle on the side of the block and in turn activate it In other words the ventricles will be activated one after the other instead of simultaneously in series instead of in parallel

It is worth mentioning that we shall encounter four conditions in which the ventricles are activated successively instead of simultaneously—bundle branch block ectopic (premature) ventricular beats paroxysmal ventricular tachycardia which after all is simply a rapid succession of ectopic ventricular beats and idioventricular rhythm originating in an ectopic ventricular pacemaker There is therefore in these four conditions a marked fundamental similarity in the bizarre pattern produced in each case there is prolongation of the QRS interval and the ST segment tends to slope off in the direction opposite to the main QRS deflection An isolated premature ventricular beat a single complex abstracted from a run of ventricular tachy

cardia and another from an example of idioventricular rhythm are illustrated side by side with a single complex from a tracing of bundle branch block in figure 34 The similarity of pattern common to all is evident at a glance

In bundle branch block the QRS interval is prolonged to 0 12 second or more and it tends to be more prolonged in left than in right

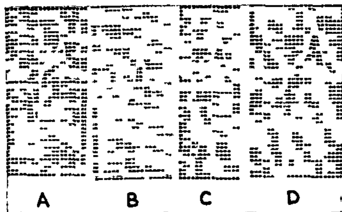


Fig 34 Comparison of patterns of (A) bundle branch block (B) ectopic ventricular beat (C) ventricular tachycardia and (D) idioventricular rhythm originating in a low ventricular pacemaker Note that each pattern has in common 1) prolonged QRS interval and 2) ST segment sloping off to T wave in direction opposite to main QRS deflection

branch block To determine whether right or left branch is affected the intrinsicoid deflections over right and left ventricles should be scrutinized When the left branch is blocked the impulse reaches the right ventricle punctually but it is late in activating the left ventricle The intrinsicoid deflection over the right ventricle (e g in V_1) therefore begins on time whereas over the left ventricle (e g in V_6) this deflection is much delayed (fig 35 A) On the other hand when it is the right branch that is blocked just the reverse occurs—the intrinsicoid deflection is on time in left ventricular leads but is late over the right ventricle (fig 35 B) The QRS complex over the right ventricle

Fig 35 **Bundle branch block (A) left and (B) right The important leads to study in BBB are 1 and 3 V_1 and V_6 (see text)**

often becomes M shaped in right bundle branch block (fig 36 B) while wide S waves appear over the left ventricle

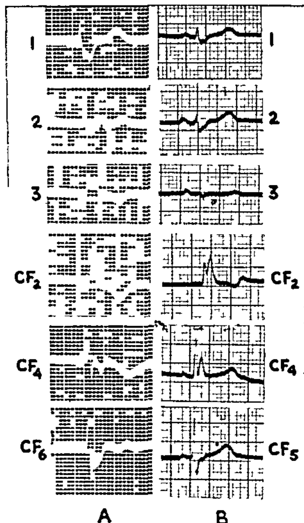


Fig 36 Right bundle branch block Note wide S_1 late intrinsicoid deflections in CF_2 and early down strokes in CF_4 and CF_6

Characteristic though less reliable changes also occur in the standard limb leads Left bundle branch block usually produces marked left axis deviation in these leads (figs 35 A and 37) Right bundle branch block on the other hand produces a prominent wide S wave in lead 1 If this S wave is sufficiently deep and the QRS complex in lead 3 is upright frank right axis deviation may be produced (figs 36 A and 38) this is the uncommon type of right bundle

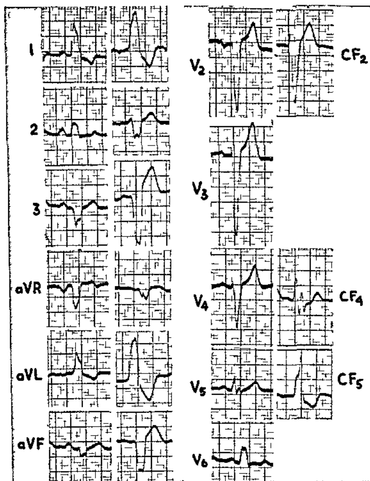


FIG 37 Two examples of **left bundle branch block** Note left axis deviation and late intrinsicoid deflections over left ventricle

branch block A much more common type is sometimes known as Wilson block, here a tall slender R wave precedes and exceeds in amplitude the wide S wave in lead 1 (fig 35 B)

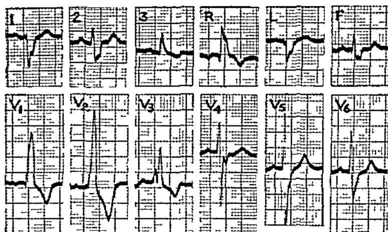


Fig 38 Right bundle branch block with marked right axis deviation—the uncommon type of RBBB

Changes in the standard leads however are very variable Indeed on the basis of such changes the patterns of left bundle branch block have been subdivided into four types and those of right bundle branch block into no less than seven It is quite unnecessary to become familiar with these several types The two diagnostic principles that must be borne in mind are 1) that the final analysis of bundle branch block rests with the precordial leads and 2) that the standard leads show a great variety of patterns Just as left axis deviation is not a sine qua non of left ventricular hypertrophy (page 52) so it is not invariably found in left bundle branch block It is not rare to see normal axis (fig 39) and it is even possible to have right axis devia

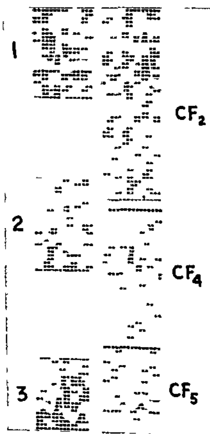


FIG 39 **Left bundle branch block** The axis is normal but the late intrinsicoid deflection over the left ventricle (CF_5) is decisive

tion if the left block affects a vertical heart (fig 40) Similarly right bundle branch block may be associated with marked left axis deviation if the heart is horizontal (fig 41) This is not infrequently seen in hypertensive hearts when right bundle branch block is superimposed on the pattern of left ventricular hypertrophy

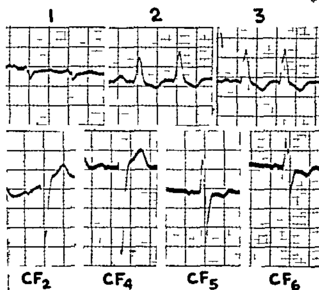


Fig 40 **Left bundle branch block** There is right axis deviation but the late intrinsicoid deflection over the left ventricle (CF_6) is decisive

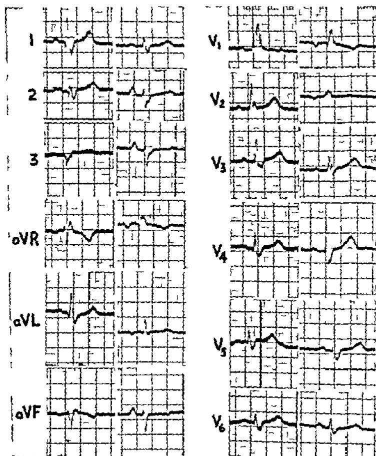


FIG 41 **Right bundle branch block** Two examples with left axis deviation A wide S_1 is present in each case intrinsicoid deflections are late over the right ventricle (V_1 and V_2) but early over the left

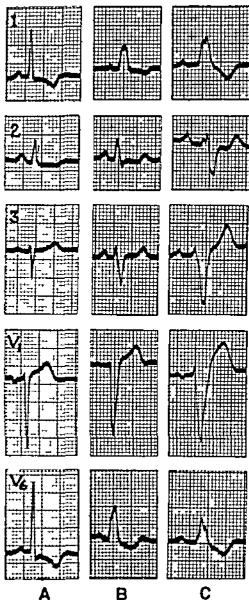


Fig 42 Left ventricular hypertrophy and strain (A) incomplete left bundle branch block (B) and complete left bundle branch block (C) compared

As intimated earlier the pattern of bundle branch block can be summarized as an exaggeration of the pattern of ventricular strain Compare A B and C in figure 42 The main differences are that the QRS interval is longer in block the intrinsicoid deflection over the blocked ventricle is correspondingly later and the ST T changes are more pronounced The QRS deflections in block however are often of lower voltage and are more likely to show definite notching than in ventricular hypertrophy and strain One further important detail should be noted whereas the normal Q waves over the left ventricle may be present or exaggerated in ventricular hypertrophy in left bundle branch block these normal Q waves are absent This is because as the left branch is blocked no impulses reach and traverse the early septal twig arising from that branch—the septum is entirely activated from its right side in left bundle branch block Figure 43 illustrates how left ventricular hypertrophy and strain sometimes progresses to left bundle branch block in the same patient

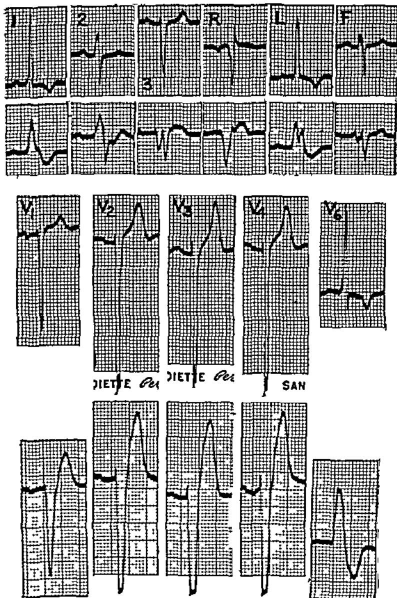


FIG 43 Two tracings from same patient taken two years apart First tracing (first and third rows) shows **left ventricular hypertrophy and strain** second tracing (second and fourth rows) shows fully developed **left bundle branch block**

It is distinctly arbitrary always to be dogmatic in differentiating ventricular hypertrophy from bundle branch block on the basis of QRS duration alone Proved cases of bundle branch block have shown QRS intervals of not more than 0 10 second, on the other hand undoubted cases of pure hypertrophy have QRS intervals of 0 12 second The designation **incomplete bundle branch block** has been assigned to those patterns whose QRS intervals place them in the no man s land of 0 10 to 0 12 second with a left bundle branch block pattern (fig 42 B) or 0 09 to 0 10 second with a right block pattern (fig 44) The term **arborization block** has been applied to the combination of bundle branch block with extremely low voltage in the limb leads

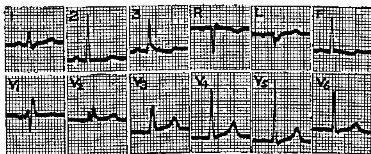


Fig 44 **Incomplete right bundle branch block** Note the salient features of the right bundle branch block pattern—wide S in lead 1 with late intrinsicoid deflection in V —but with QRS duration of only 0 10 sec

Left bundle branch block statistically at any rate carries with it a less favorable prognosis than right (2 3 6) It is obvious however that the ultimate outlook depends not on the conduction disturbance per se but on the disease that is causing it Therefore in any given instance of coronary disease causing bundle branch block the prognosis should obviously be based not on which bundle the disease process has happened to affect but on one s estimate of the severity of the underlying coronary disturbance **Intermittent bundle branch block** i e prolonged QRS complexes present at times but not at others (figs 45 and 46) presumably carries the same import as permanent block and probably represents a transition stage before permanent block is established

Left and right bundle branch blocks occur with about the same frequency (3) Coronary disease is much the commonest cause of persistent bundle branch block Other causes are rheumatic disease syphilis trauma tumors and congenital lesions Transient bundle branch block may occur in acute heart failure acute myocardial infarction acute coronary insufficiency and acute infections or may result from digitalis or quinidine intoxication

Salient features of bundle branch block

Leads	Left bundle branch block	Right bundle branch block
V_1	early intrinsicoid	late intrinsicoid M shaped QRS
V_6	late intrinsicoid no Q waves	early intrinsicoid
Standard leads	left axis deviation usually	wide S_1 with or without axis deviation

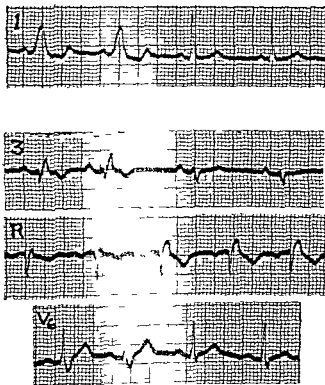


FIG 45 Intermittent bu[illegible]lle branch block The first strip is lead 1 from one patie[illegible] three leads are from another patient Note the variat[illegible] QRS [illegible]ttern and duration In the first strip the first two beats [illegible] intr[illegible]tricular block the last two are of normal duration a[illegible] each [illegible] the other leads it can also be seen that some QRS c[illegible]les [illegible] long[illegible]d while others are of normal duration

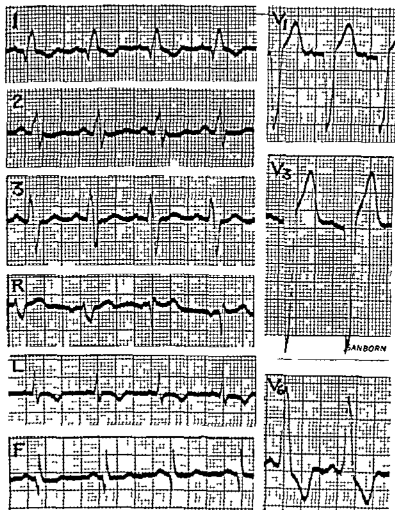


Fig 46 **Intermittent intraventricular block** Intraventricular block presumably of left bundle branch type prevails in standard limb leads during aVR relatively normal intraventricular conduction appears and continues through the aV leads block reappears in V_1 and continues throughout the precordial lead

THE WOLFF PARKINSON WHITE SYNDROME

This purely electrocardiographic syndrome (9) is conveniently considered here because the prolonged QRS that characterizes it gives it a superficial resemblance to the bundle branch block pattern. It has been called pseudo bundle branch block.

The pattern consists of a shortened P R interval followed by a lengthened QRS interval. The QRS is as much longer than normal as the P R interval is shorter than normal. The P R interval usually measures 0 10 second or less and the P wave is normal. The upstroke of the QRS in lead 1 is usually heavily slurred producing the so called delta wave (fig 47 A).

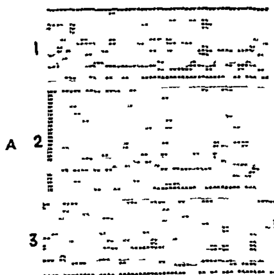


Fig 47 A Wolff Parkinson White syndrome Note short P R interval prolonged QRS interval and delta waves (slurred upstrokes) in lead 1

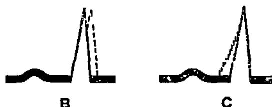


Fig 47 B and C

B Bundle branch block and C W P W syndrome compared diagrammatically

If the pattern of bundle branch block is superimposed on a normal P QRS sequence we get the composite picture indicated in figure 47 B the blocked ventricle is activated late so that the superadded tracing is tacked on to the downslope of the normal QRS leaving the P R interval untouched In the W P W syndrome the superadded tracing is attached in front of the normal QRS thus shortening the P R interval by as much as it lengthens the QRS (fig 47 C) In bundle branch block we know that the QRS is prolonged because one ventricle is activated late it seems reasonable to assume that the W P W syndrome results when one ventricle (or part of a ventricle) is activated abnormally early This phenomenon is called **pre excitation**

The explanation is generally accepted and has indeed been proved but the exact mechanism of early activation of one ventricle is in dispute It has been widely believed that accelerated conduction to one ventricle is effected through an accessory bundle (bundle of Kent) which by passes the A V node and bundle and so avoids the pause which normally occurs when the impulse arrives at the A V junctional tissues Prinzmetal (8) has recently presented evidence that the asynchronous activation of the ventricles in this syndrome is due to differential transmission of the impulse at the A V node itself in other words most of the impulse suffers its normal delay at the A V node while part of the impulse speeds ahead to its ventricular destination He has suggested the term **accelerated conduction syndrome** as a descriptive alternative for Wolff Parkinson White syndrome

This syndrome is a relatively benign condition It occurs mainly in individuals with no sign of heart disease whose only other cardiac peculiarity is a tendency to paroxysmal atrial tachycardia At times however this anomaly of conduction appears to result from

serious heart disease and therefore cannot be said to be invariably associated with a benign prognosis It affects males predominantly and is found in all age groups

REFERENCES

BUNDLE BRANCH BLOCK

1 DeForest R F Four cases of benign left bundle branch block in the same family Am Heart J 1956 51 398
2 Johnson R I et al Prognosis in bundle branch block II Factors influencing the survival period in left bundle branch block Am Heart J 1951 41 225
3 Messer A L et al Prognosis in bundle branch block III A comparison of right and left bundle branch block with a note on the relative incidence of each Am Heart J 1951 41 239
4 Myers G B The form of the QRS complex in bundle branch block and in anterolateral infarction Am Heart J 1950 39 817
5 Papp C and Smith K S The changing electrocardiogram in Wilson block Circulation 1955 11 53
6 Shreenivas et al Prognosis in bundle branch block I Factors influencing the survival period in right bundle branch block Am Heart J 1950 40 891
7 Vazifdar J P and Levine S A Benign bundle branch block Arch Int Med 1952 89 568

WOLFF PARKINSON WHITE SYNDROME

8 Prinzmetal M et al Accelerated conduction *The Wolff Parkinson White Syndrome and Related Conditions* Grune and Stratton New York 1952
9 Wolff L Syndrome of short P R interval with abnormal QRS complexes and paroxysmal tachycardia (Wolff Parkinson White syndrome) Circulation 1954 10 282

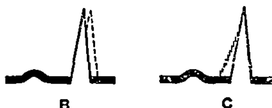


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5 Rapp C and Smith K S The changing electrocardiogram in [illegible] block Circulation 1955 11 53
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7 Vazifdar J P and Levine S A Benign bundle branch block Arch [illegible] Med 1952 89 568

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9 Wolff L Syndrome of short P R interval with abnormal QRS complexes and paroxysmal tachycardia (Wolff Parkinson White syndrome) Circulation 1954 10 282

More Practical Points

The technician is not expected to learn how to interpret the tracing but she should be told certain useful points that will make her work more intelligent and more interesting She should watch the tracing come out of the machine with a trained alert eye and she should be given the following practical instructions

Notice carefully the pattern of these four leads—

1 *Lead 1* If the complexes are inverted check your arm electrodes—they are almost certainly reversed (fig 143 B page 197)

2 *Lead 3* If the first deflection of the ventricular complex is downward (a Q wave) tell the patient to take a deep breath and hold it for a few heart beats (figs 110 and 111 page 152) This will help to distinguish between important and unimportant Q waves

3 *Lead* V_1 If the ventricular complex shows a tall upright (R) wave instead of the usual deep downstroke take a lead or two further to the right (V_{3R} V_{4R}) to try and get a right ventricular lead with the major deflection downward

4 *Lead* V_4 If the ventricular complex shows a deep down stroke (S wave) instead of the usual tall upright wave take a lead or two further to the left (V_7 V_8) to try and get a left ventricular lead pattern with the major deflection upward

8

The Arrhythmias Ventricular Arrhythmias

Disturbances of rhythm are most conveniently divided into a) supraventricular and b) ventricular This corresponds with a simple electrocardiographic difference—arrhythmias originating in the atrium or A V node (supraventricular) are characterized by normal QRS complexes while ventricular arrhythmias produce bizarre QRST complexes with prolonged QRS interval Exceptions rarely occur Therefore the golden rule in interpreting arrhythmias is **look first at the QRS interval** If this interval is normal the arrhythmia is of supraventricular origin if it is prolonged the arrhythmia is *probably* of ventricular origin

With a prolonged interval we can only say *probably* ventricular because there is always the possibility that intraventricular block is coincident with a supraventricular disturbance of rhythm Such a block may have been present before the supraventricular arrhythmia began or again the strain put upon ventricular conduction by an arrhythmic tachycardia may have induced a temporary intraventricular block The pattern of intraventricular block plus supraventricular tachycardia may be virtually indistinguishable from ventricular tachycardia Bearing this exception in mind when a tachycardia is seen with bizarre prolonged QRS complexes it is best diagnosed as a ventricular tachycardia till proved otherwise

Two other practical justifications for the subdivision of arrhythmias into supraventricular and ventricular are 1) it is sometimes impossible to distinguish between atrial and A V nodal tachycardias and 2) the treatment of both supraventricular tachycardias is the same

Arrhythmias may be recognizable in any lead but as a general rule the most satisfactory leads for diagnosis are leads 2 3 and V_1

VENTRICULAR ARRHYTHMIAS

Four ventricular arrhythmias are generally described—ectopic beats tachycardia flutter and fibrillation

The terms **ectopic beat premature beat** and **extrasystole** are virtually synonymous All ectopic beats however are not premature For example the ventricular response in atrial fibrillation may be interrupted by an ectopic ventricular beat but as the time of the next ventricular response during atrial fibrillation cannot be predicted it is obviously ridiculous to call such a beat premature For this reason ectopic beat is here preferred as the generic term though premature beat and extrasystole will be freely used when their use seems appropriate

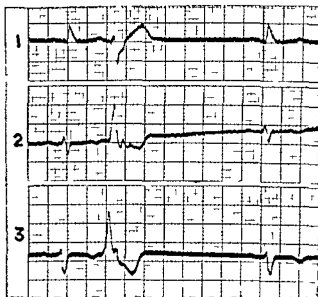


Fig 48 **Premature ventricular beats** The second beat in each lead is an ectopic (premature) ventricular beat Notice that while the dominant complexes show left axis deviation the ectopic beats show *right* axis shift They therefore arise in the *left* ventricle (see pages 36–3)

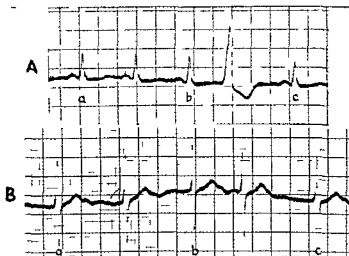


FIG 49 Comparison of interval following **premature ventricular beat** (A) and that following a **supraventricular premature beat** (B) see text

ECTOPIC VENTRICULAR BEATS

The isolated ectopic ventricular beat is perhaps the most easily recognized of all cardiographic aberrations It sticks out grotesquely like a sore thumb Several are shown in the strips in figures 48 to 53 It can be seen that the ectopic impulses have produced distorted QRST complexes all of which have certain characteristics in common 1) they anticipate the next normal impulse (i e they are premature) 2) the QRS intervals are prolonged 3) the ST segments slope away in the direction opposite to the main QRS deflections 4) following each premature beat there is a relatively long pause until the next sinus impulse in due time initiates the next cardiac cycle this pause is as much longer than the normal cardiac cycle as the ectopic beat was premature Thus the interval from the beat preceding the ectopic beat to that following it is exactly equal to two cardiac cycles i e in strip A in figure 49 the interval from b to c equals that from a to b The pause following the premature beat is therefore **fully compensatory** in contrast with the interval that follows a supraventricular beat (strip B in fig 49) Careful measurement in strip B will show that the interval from b to c *is not quite equal to that from a to b*

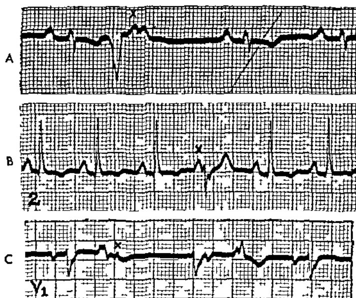


Fig. 50 Relationship of P waves to **premature ventricular beats** In A the ventricular beat is very premature and the P wave (x) deforms the ST segment In B the ectopic beat is only slightly premature and the P wave (x) precedes it In C the first premature beat is followed by a retrograde P wave (x) retrograde conduction to the atria is not observed following the second premature beat

The P wave is usually lost in a premature ventricular beat because the QRS complex is usually sufficiently premature to swamp it Occasionally it may be seen as a notch or splinter slightly deforming some part of the QRS complex or even the T wave (fig 50 A) On the other hand if the ectopic beat is only slightly premature the normal P wave may have time to put in an appearance shortly before the abnormal QRS (fig 50 B) Not rarely the ectopic impulse from the ventricle spreads backwards into the atria inscribing a **retrograde P wave** which replaces the sinus P wave that was otherwise expected (2) (fig 50 C) Occasionally the ectopic beat is so premature that the next sinus impulse finds the ventricle sufficiently recovered to respond In such circumstances the premature beat is sandwiched in between two normally paced sinus beats and is known as an **interpolated beat** (fig 51 B)

When every other beat is a premature beat (fig 52) the rhythm is described as **bigeminy** (pulsus bigeminus, bigeminal rhythm)

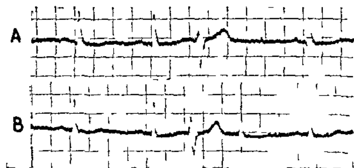


FIG 51 **Premature ventricular beats** A The premature beat is as usual followed by compensatory pause B The premature beat is sandwiched in between two normal sinus beats—an **interpolated ventricular premature beat**

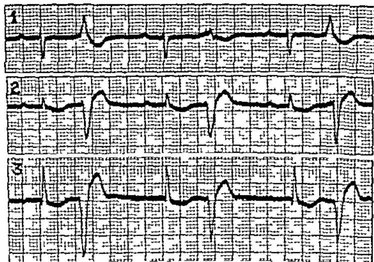


FIG 52 **Bigeminal rhythm** In each lead every alternate beat is a premature ventricular beat In this patient the dysrhythmia was due to digitalis intoxication

or **coupled beats** If every third beat is a premature beat (fig 53 A), or if each normal beat is followed by a pair of premature beats (fig 53 B and C), **trigeminy (pulsus trigeminus, trigeminal rhythm)** is produced

Ectopic ventricular beats are generally regarded as a normal finding unless they are very numerous or arise from more than one focus **(multifocal or multiform ectopic beats)** (fig 53 B and C) Even bigeminal rhythm however is sometimes seen in an otherwise normal heart When both ventricular and atrial ectopic beats are observed

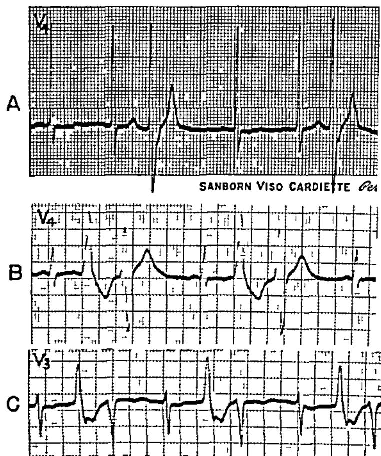


FIG 53 **Trigeminal rhythm two forms** In A every third beat is a ventricular premature beat in B and C each sinus beat is followed by a pair of ventricular premature beats

in the same heart it is generally considered evidence of cardiac disease (1)

> Salient features of ectopic ventricular beats
> 1 bizarre premature QRST complex with prolonged QRS interval and ST segment sloping off in direction opposite to main QRS deflection
> 2 followed by *fully* compensatory pause (unless interpolated)
> 3 P wave usually lost (submerged in ventricular complex)

PARASYSTOLE

Premature ventricular beats tend to occur during the supernormal recovery phase of the ventricle which corresponds with the U wave in the electrocardiogram During this phase of supernormal excitability the ventricles can be activated by stimuli which are otherwise sub threshold Premature beats therefore tend to bear a fixed relationship to the preceding beat this fixed relationship is known as **fixed coupling** and the ectopic beat is thought to be in some way causally related to the preceding beat

Sometimes however an autonomous ectopic focus in the ventricle fires off regular impulses at its own independent rate and whenever these impulses arise at a time outside the refractory period an ectopic beat results This phenomenon of a parallel pacemaker competing for control is known as **parasystole** it is to be suspected whenever (1) the ectopic beats show a varying time relationship to the preceding beats and (2) the intervals between consecutive ectopic beats are all equal to or are multiples of the shortest inter ectopic interval observed (fig 54)



FIG 54 Possible **parasystole** Every second beat is premature but the interval between premature beat and preceding beat is variable i e there is no fixed coupling The inter ectopic interval (distance between consecutive premature beats) however is constant These two features are compatible with but not diagnostic of parasystole Careful measurement in a much longer strip would be necessary for precise diagnosis

PAROXYSMAL VENTRICULAR TACHYCARDIA

Ventricular tachycardia is merely a run of rapidly repeated premature beats It is recognized by the appearance of bizarre prolonged QRS complexes recurring at a rapid rate They are more or less regular but this is the least regular of the so called regular arrhythmias (see table on page 126) The P waves are frequently lost though they may sometimes be recognized as notches occurring at a slower rate and usually in no constant relation hip to the ventricular complexes (figs 55 and 56) In fact, identification of unrelated P waves is the only certain means of recognizing ventricular tachycardia



Fig 55 **Ventricular tachycardia** at relatively low rate of 120 Independent P wave are clearly seen at slower rate (92)

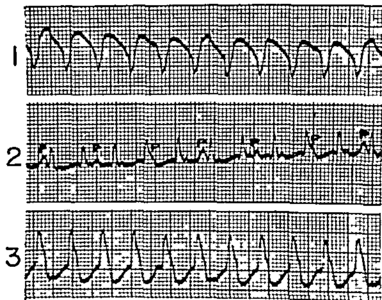


Fig 56 **Ventricular tachycardia** at rate 180 Typical pattern in leads 1 and 3 but the diagnosis is conclusively established by the visibly independent P waves in lead 2 at rate 110

—if such P waves are not seen there is always the possibility that a supraventricular tachycardia is associated with intraventricular block

Thus it is sometimes impossible to differentiate true ventricular tachycardia from a supraventricular tachycardia complicated by intraventricular block The combination of supraventricular tachycardia with intraventricular block may be suspected however if the patient is known to have been subject to supraventricular arrhythmias i e if premature atrial beats or atrial tachycardia for example have been observed in previous tracings It may be strongly suspected if the patient had a pre existing bundle branch block and with the onset of the tachycardia the pattern of the QRST complexes has remained unchanged It can be diagnosed with certainty only in the event that abnormal P waves are seen in constant relationship to each abnormal QRS complex

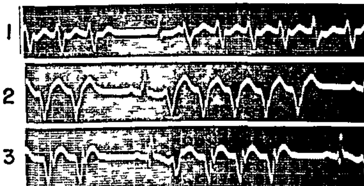


Fig 57 Ventricular tachycardia Short runs of ventricular tachycardia at rate of 115

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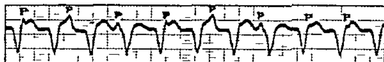


Fig 55 Ventricular tachycardia at relatively slow rate of 1?0 Independent P waves are clearly seen at slower rate (9?)

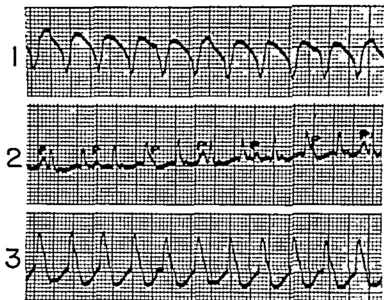


Fig 56 Ventricular tachycardia at rate 185 Typical pattern in leads 1 and 3 but the diagnosis is conclusively established by the visibly independent P waves in lead 2 at rate 110



FIG 60 Two serious forms of ventricular tachycardia usually presaging ventricular fibrillation and death A **Bidirectional ventricular tachycardia** B **Multifocal ventricular tachycardia** (sometimes called **chaotic heart action**)

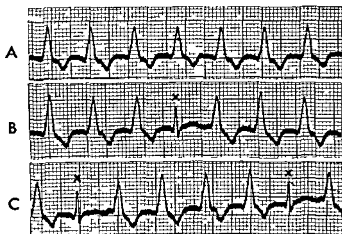


FIG 61 **Ventricular tachycardia** A Typical form at relatively slow rate of 130 B and C form a continuous strip in which occasional atrial impulses are conducted (x)

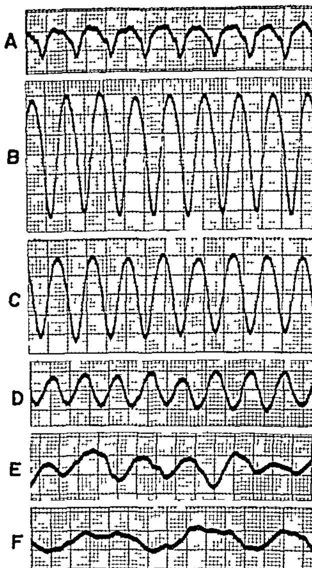


Fig 62 **The dying heart** *Strips from lead 2 taken approximately 1 minute apart* and illustrating the transition from ventricular tachycardia through flutter to fibrillation **A Ventricular tachycardia** **B and C Ventricular flutter** **D** Intermediate stage between flutter and fibrillation **E and F Ventricular fibrillation**

ventricular rate is relatively slow and indicates that an impulse from the independently beating atria happening to arrive at an opportune moment when the ventricles were no longer refractory has been conducted

Ventricular tachycardia is less common than atrial tachycardia but it is much more serious It is usually a sign of grave heart disease and is a not uncommon prelude to ventricular fibrillation **Ventricular flutter** is the term given by some authorities to a rapid ventricular tachycardia giving a slightly modified pattern in the electrocardiogram (fig 62 B and C) Nothing is gained in separating it from ventricular tachycardia

Ventricular fibrillation is usually a terminal event and is therefore of only academic importance rarely transient bout may be responsible for Adams Stokes attacks It is easily recognized in the electrocardiogram by the complete absence of properly formed ventricular complexes—the baseline is seen to waver unevenly with no attempt at forming clearcut QRS deflections (fig 62 E and F)

REFERENCES

1 Huppert V F and Berliner K Auricular premature systoles occurring at rapid heart rates Bull N Y M d Coll 1951 14 23
2 Kistin A and Landowne M Retrograde conduction from premature ventricular contraction a common occurrence in the human heart Circulation 1951 3 738
3 Mack I and Langendorf R Factors influencing the time of appearance of premature systoles Circulation 1950 1 910
4 Pick A Parasystole Circulation 1953 8 24
5 Scherf D and Schott A *Extrasystoles and Allied Arrhythmias* Grune and Stratton New York 1953

9

The Arrhythmias Atrial Arrhythmias

SINUS RHYTHMS

Before dealing with the atrial arrhythmias proper a few words should be said about the sinus (S A) rhythms In all of these there is normal impulse formation at the S A node and normal spread of the impulse from here to the A V node The hallmark of all sinus rhythms is therefore a normal P wave

The normal rate of impulse formation by the sinus node is generally accepted as 60 to 100 per minute Above 100 the rhythm is called **sinus tachycardia** (fig 63 A) below 60 **sinus bradycardia** (fig 63 B) Sinus tachycardia results from exercise eating emotion pain hemorrhage shock fever thyrotoxicosis and infections it is a common reaction to heart disease and heart failure per se and may be caused by many drugs such as caffeine nicotine adrenaline atropine amyl nitrite and quinidine Sinus bradycardia is seen as a normal variation especially in well trained athletes whose heart rates may be in the thirties at rest— and often not much more with exertion it is a physiologic reaction to sleep fright carotid sinus massage or ocular pressure and it may also result from disease processes such as obstructive jaundice (effect of bile salts on sinus node) glaucoma (oculocardiac reflex) carotid sinus sensitivity and increased intracranial pressure it is often seen in convalescence and as a result of digitalis therapy

When the sinus node forms impulses irregularly, we have **sinus arrhythmia** (fig 63 C) This is of two varieties one that waxes and wanes with the phases of respiration the heart accelerating with inspiration and slowing with expiration and a less common type in

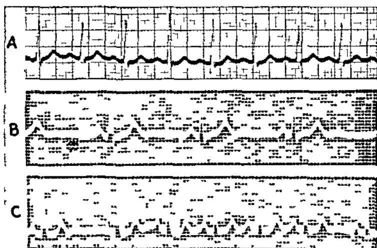


FIG 63 A Sinus tachycardia B Sinus bradycardia C Sinus arrhythmia

which the changes of rate bear no relationship to the phases of respiration Sinus arrhythmia is a perfectly normal finding but it may on occasion produce such marked irregularity that it can be confused clinically with other more important arrhythmias

THE SUPRAVENTRICULAR ARRHYTHMIAS

These arrhythmias arise from either a) an irritable ectopic focus in the atrium or b) the A V node They are all characterized (unless they are complicated by coincident intraventricular block) by normal QRS duration

ATRIAL ARRHYTHMIAS

Four atrial arrhythmias are generally described ectopic beats paroxysmal tachycardia flutter and fibrillation For some time it ha been generally considered that ectopic beats and tachycardia have a common mechanism—discharge from an irritable ectopic focus in the atrium—but that flutter and fibrillation arise by a different mechanism namely circus movement Recently Prinzmetal (5) has presented evidence for the *unitary nature* of the atrial arrhythmias demonstrating that all four arrhythmias can and probably do arise from an ectopic focus and that circus movement is almost certainly *not* the underlying mechanism of any of these arrhythmias

According to Prinzmetal's thesis, all four atrial arrhythmias have the same underlying mechanism: more or less frequent discharge of impulses from an ectopic focus in one of the atria. It appears that the most important factor in determining which arrhythmia develops is the *rate* of discharge from this focus. If the rate of discharge is very rapid, fibrillation results; if the rate of discharge is 300 or so, flutter is produced. If the rate is in the neighborhood of 200, paroxysmal tachycardia occurs. With rates of 100 or less, more or less frequent premature beats arise.

This simple concept has much to recommend it besides the impressive factual evidence that has been accumulated in its support. It leaves a few minor points unexplained but it is overwhelmingly better and more thoroughly documented than the theory of circus movement which held almost undisputed sway for a quarter of a century.

If we accept this unitary concept, there is no fundamental difference between tachycardia and flutter; this point will be further discussed on pages 103–4.

ECTOPIC ATRIAL BEATS

When an ectopic focus (i.e., a point in the atria other than the S-A node) initiates an impulse, this impulse obviously travels across the atria by an unusual path and therefore creates a distorted, often

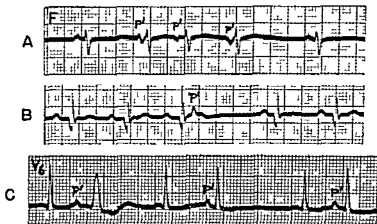


FIG. 64. **Ectopic atrial beats** labelled P'. A. Three consecutive premature atrial beats are shown, each arising from a different atrial focus (three differently shaped P' waves). B. After the third sinus beat a very early premature beat occurs; finding the ventricles refractory, it is not conducted. C. **Atrial bigeminy**: every other beat is a premature atrial beat, the most premature

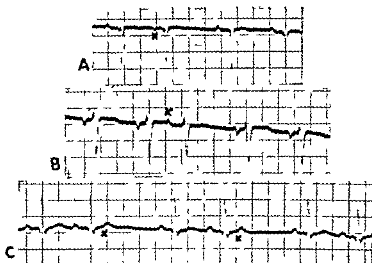


FIG 64 **Premature atrial beats** A and B illustrate abnormal premature P waves (X) followed by normal QRST sequences C Ectopic P waves are seen as notches on the T waves (X) and are not followed by ventricular complexes These are easily identifiable as abnormal P waves since the T waves of other cycles are smooth and unnotched

inverted P wave When this impulse arrives at the A V node it will proceed down the orthodox A V conducting paths like any other supraventricular impulse and if the ventricle is not refractory will spread through and normally activate the ventricular myocardium

We therefore recognize an ectopic atrial beat by the premature abnormal P wave Emphasis has been laid on *inversion* of the P wave It is true that often the ectopic P wave is inverted where it should be upright (leads 1 2 and aVF) and upright where it should be inverted (aVR) but this is not necessarily so and the important feature of the ectopic P is simply that its form *differs* from that of the normal P in the lead in question

Usually the ventricles respond to the premature atrial impulse and a normal QRST sequence is inscribed (figs 64 A and C 65 A and B) If however the atrial impulse is very premature so that it finds the ventricle still refractory the ectopic P wave will not be followed by a ventricular complex—a non conducted atrial premature beat (figs 64 B and C C) At times the impulse may find the ventricles only partly refractory and so have to travel through them by an aberrant path this results in an abnormal QRST sequence and the phenomenon is known as **aberrant ventricular conduction** (31 or **ventricular aberration** (fig 64 C)

The interval following a premature atrial beat is usually somewhat longer than the normal cardiac cycle, but is not so long as to be *fully* compensatory (page 83) The difference in the time relationships between atrial and ventricular premature beats was illustrated in figure 49 page 83

Salient features of ectopic atrial beats
1 abnormal often inverted premature P wave
2 normal QRST
3 ensuing interval is about equal to or slightly longer than the normal cardiac cycle

PAROXYSMAL ATRIAL TACHYCARDIA

This arrhythmia is best thought of as a run of rapidly repeated premature beats It is therefore characterized by normal QRS complexes appearing at a rapid rate usually between 160 and 250 per minute Their spacing is usually perfectly regular though slight irregularity sometimes occurs Theoretically an abnormal P wave precedes each QRS complex but in practice the rate is usually so rapid that the P wave is merged with the preceding T wave and is indiscernible The arrhythmia is therefore diagnosed by the occurrence of normal QRS complexes at high speed and considerable regularity If the interpreter waits for the appearance of abnormal P waves he will diagnose only a small proportion of cases It is often impossible to distinguish between atrial and A V nodal tachycardia it is frequently wisest to commit oneself to the diagnosis supraventricular tachycardia For a general discussion of this differentiation see pages 126–7

Secondary changes in the ST T segment may occur Any tachycardia shortens diastole and therefore curtails coronary blood flow, if the coronaries are already diseased and sometimes even if they are normal ST T changes characteristic of coronary insufficiency may develop These changes consist of depression of the ST segment with inversion of the T wave and are well shown in figure 66 It is important to note that ST and T wave changes of this sort may persist for hours or days after the paroxysm of tachycardia has ceased (**post tachycardia syndrome**)

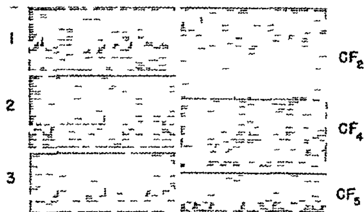


Fig 66 **Atrial tachycardia** Rate 255 Note normal QRS interval and ST depression in all leads

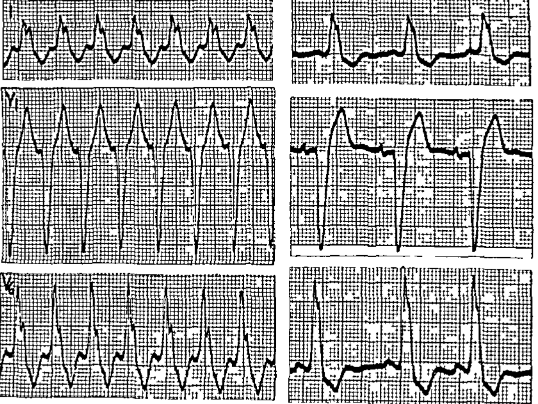


Fig 67 **Supraventricular tachycardia** associated with **intraventricular block** A The tracing is indistinguishable from ventricular tachycardia B When sinus rhythm is later restored the QRST pattern remains virtually unchanged proving that a supraventricular rhythm was present in A

It has already been mentioned that atrial arrhythmias may be complicated by intraventricular block (fig 67) In such circumstances the QRS complexes will obviously be prolonged and this combination can only be differentiated from ventricular tachycardia if the abnormal P waves happen to be discernible

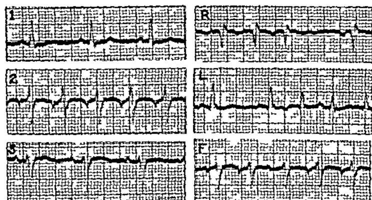
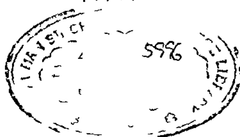


FIG 68 **Paroxysmal atrial tachycardia** Tracing shows onset and offsets of short bursts of atrial tachycardia Leads 1 and 3 show normal sinus rhythm lead 2 shows atrial tachycardia rate 144 lead aVF also shows the tachycardia but the third beat arises from a separate focus lead aVR shows the last three beats of a paroxysm followed by a sinus beat aVL shows two sinus beats followed by the first three beats of a paroxysm of tachycardia

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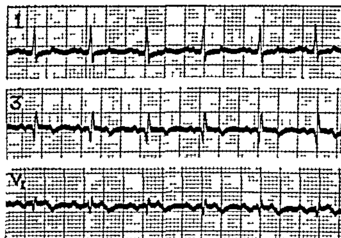


Fig 69 A

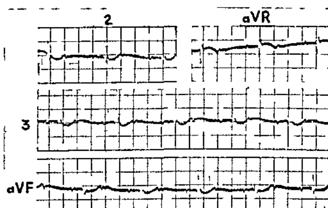


Fig 69 B

Fig 69 Two examples of **atrial tachycardia** with 2 1 A V block This rhythm would be called atrial flutter by some authorities because of the presence of A V block (see p 103)

Salient features of paroxysmal atrial tachycardia
1 rapid (150–250) regular normal QRS complexes
2 abnormal P waves preceding QRS (rarely discernible)
3 ST T depressions frequently seen

ATRIAL FLUTTER

Flutter versus tachycardia

It has already been pointed out that according to Prinzmetal s evidence the main difference between atrial tachycardia and flutter is that flutter represents a faster discharge from the ectopic focus in the atrium In the past flutter has been arbitrarily separated from tachycardia on the basis of a) atrial rate b) presence or absence of A V block or c) the shape of the atrial complexes

a) An *atrial rate* of 250 has been set as the dividing line between tachycardia and flutter A distinction as discretionary as a numerical boundary cannot be defended single bouts of tachycardia have been observed in which the atrial rate has wandered on both sides of this boundary within a single uninterrupted attack

b) The *presence of A V block* is used by some as a diagnostic criterion of flutter (see fig 69) But it is clear that the rate at which the ventricles fail to keep pace with accelerated atria is determined by the efficiency of the individual A V conducting tissues and not by the atrial mechanism Block will not develop in one heart till a rate of say 250 is attained whereas it may develop at 200 in another By the criterion of block the rhythm will still be called paroxysmal tachycardia at 240 in the first heart while at 210 in the second heart it will already be flutter—simply because the second heart has a less efficient A V conducting system

c) The *form of the P waves* is perhaps the most characteristic of the traditional criteria for diagnosing flutter As far as we know at present the shape and direction of abnormal atrial waves depend mainly on two factors the *part* of the atrium in which the ectopic focus is situated, and the *rate* of discharge from that focus Apparently the faster the rate of discharge the more likely is the atrial wave to become distorted into the form that we have come to regard as typical of flutter This is not the whole story however for the typical con

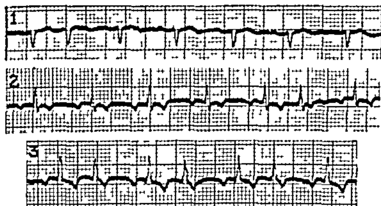


Fig 70 **Atrial tachycardia** (or flutter) with A V block The P waves are barely discernible in lead 1 and are inverted in 2 and 3 The A V block varies between 2 to 1 and 3 to 2 In lead 3 the 3 to 2 ratio is constant leaving the ventricular beats grouped in pairs

tour of flutter waves may be seen at times at an atrial rate of no more than 200 whereas the small inverted complexes usually associated with paroxysmal tachycardia may be seen at rates near 300 Furthermore these complexes may have the one form in one lead and the other in another lead in the same tracing

In view of these considerations it is clearly arbitrary and illogical to separate flutter from tachycardia on the basis of rate presence of A V block or form of the atrial complexes Opponents of the unitary concept however further point out that flutter is more common in elderly patients with heart disease whereas tachycardia is more common in the young and healthy that tachycardia more often begins in the cephalic region of the atria whereas flutter more often originates caudally and that whereas vagal stimulation frequently abolishes tachycardia it usually has little effect on flutter and may indeed convert it to fibrillation These arguments are of importance but as tachycardia and flutter cannot be clearly and exclusively defined distinctions such as these cannot with certainty be attributed to either of them It is therefore more logical to lump them together as paroxysmal tachycardia with or without A V block As this revision of nomenclature has not yet been widely accepted for the present it seems wisest to include in this section a description of the traditional pattern of flutter

Classical flutter

Typical atrial flutter is recognized by the presence of saw tooth (fig 71–74) or undulating (fig 75) atrial waves with ventricular responses following every 2nd 3rd 4th (up to 8th) atrial wave These waves have been labelled F (for flutter) wave It is important to realize that these waves do not reveal themselves with equal clarity in all leads Leads 2 and 3 usually show flutter waves most clearly while lead 1 may be particularly treacherous in giving virtually no evidence of the typical pattern (figs 71 72 74) The most common rate of flutter waves is between 300 and 320 with a 2 1 block producing therefore a ventricular rate of 150 to 160

The QRS complex as with all atrial arrhythmias is of normal duration unless intraventricular block coincidentally complicates the picture The T wave is usually dominated by the zigzagging atrial complexes so that it is not clearly identifiable but it often distorts the otherwise uniform saw tooth pattern

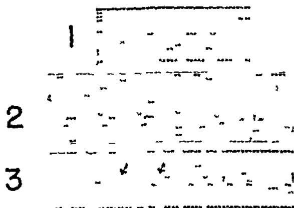


Fig 71 Atrial flutter Note saw tooth waves in leads 2 and 3 This is commonest rate of flutter (about 300) and commonest degree of A V block (2 to 1) giving a ventricular rate of about 150 Isoelectric shelf is clearly seen in lead 3 (indicated by arrows)

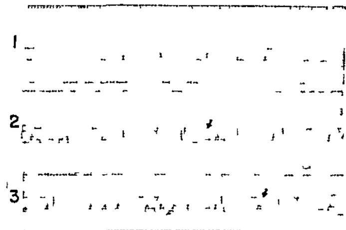


Fig 72 **Atrial flutter** In leads 2 and 3 the block is consistently 4 to 1 giving a regular ventricular rate of about 8 Block is evidently variable in lead 1 producing irregularity which could be confused with fibrillation Isoelectric shelf is clearly seen in leads 2 and 3 (indicated by arrows)

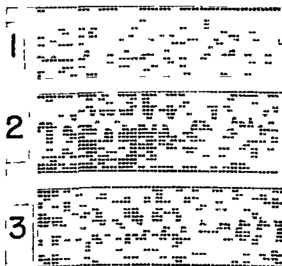


Fig 73 **Atrial flutter** In leads 2 and 3 the block varies between 2 to 1 and 4 to 1

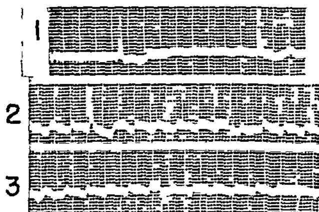


Fig 74 **Atrial flutter** The atrial rate is slow being only about 225 There is a high grade of A V block the ventricular rate being only 35 This represents a ratio between 6 and 7 to 1 The diagnosis is presumably atrial flutter with **complete A V block** and resulting **idioventricular rhythm**

Salient features of atrial flutter

1 saw tooth or undulating baseline of F waves

2 normal QRS complexes in 2 1 to 8 1 ratio

3 T waves swamped by the F wave pattern

Analysis of flutter waves

The saw tooth waves of flutter have been interestingly analyzed by Prinzmetal. He has demonstrated that the downward deflection is the abnormal P (P′) wave and is followed by an upward Ta (or T_P) wave, i.e., the wave of atrial repolarization. This rapid P′ Ta sequence is followed by a horizontal isoelectric pause before the next P′ wave begins. The length of this pause is determined by atrial rate, becoming longer as the rate slows and disappearing altogether at very rapid rates. He has termed this interval the **isoelectric shelf** and it is clearly seen in the tracings in figures 71 and 72. Figure 76 illustrates diagrammatically both the development of Ta waves and the shortening of the isoelectric shelf as the rate increases.

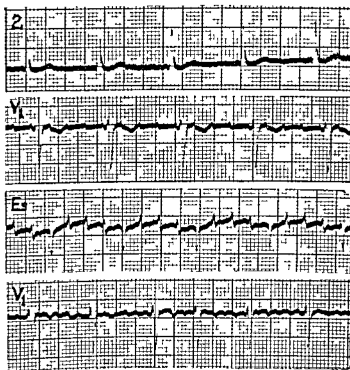
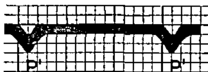
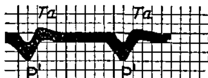
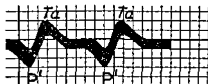


Fig. 75. **Atrial tachycardia** (or flutter) with 4 to 1 A-V block. The only routine lead that showed any sign of atrial activity was V_1. The third strip (Fs) is an esophageal lead. The fourth strip is V_1 after digitalization showing **atrial fibrillation**.

AURICULAR RATE / MIN







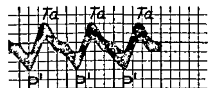




Fig. 6 Illustrating that with increase in rate 1) the Ta wave assumes greater prominence and 2) the isoelectric shelf becomes shorter Reproduced with kind permission of the publishers from *The Auricular Arrhythmias* by Myron Prinzmetal and others Charles C Thomas 1951

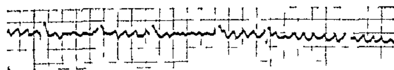


FIG A

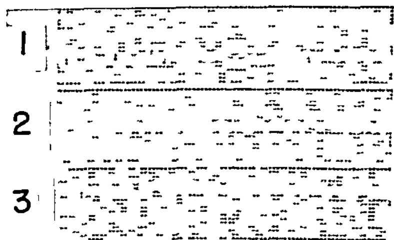


FIG B

FIG 77 **Flutter fibrillation** A The tracing shows a combination of flutter and fibrillation waves

B Note undulating flutter waves in lead 2 towards the end of this lead atrial complexes suddenly become more rapid and more erratic as fibrillation sets in

ATRIAL FIBRILLATION

When the ectopic discharge becomes excessively rapid it also becomes irregular and ineffective It no longer leaves a regular imprint on the electrocardiogram and it no longer elicits a regular ventricular response The uneven and irregular deviations in the tracing that now represent atrial activity are labelled f waves (figs 78–80) At times no sign at all of atrial activity is visible in the routine leads

Sometimes a mixture of fibrillation with flutter is seen Such a pattern is seen in figure 77 and may be called **flutter fibrillation** or **impure flutter**

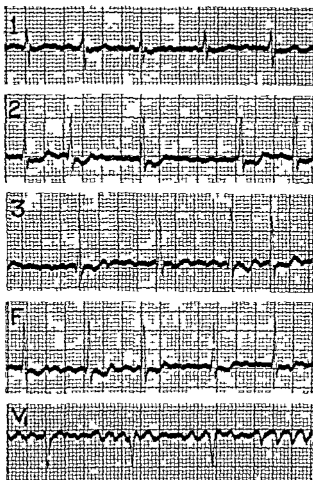


Fig. 8 **Atrial fibrillation** The typical uneven irregular f waves are visible in all leads

The irregularity of fibrillation may be clinically imitated by five other arrhythmias

1 frequent ectopic beats—ventricular atrial or A V nodal These often produce a pulse deficit Note that a pulse deficit is *not* diagnostic of atrial fibrillation
2 atrial tachycardia (flutter) with varying A V block
3 sinus rhythm with varying A V block
4 gross sinus arrhythmia
5 wandering pacemaker

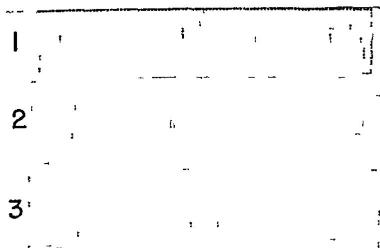


Fig 79 Atrial fibrillation with right axis deviation this combination is almost diagnostic of mitral stenosis

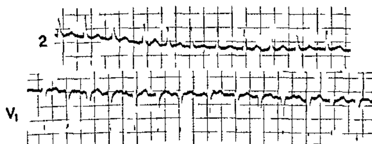


Fig 80 Atrial fibrillation with unusually regular ventricular response

The important causes of atrial fibrillation are rheumatic heart disease with mitral valve involvement coronary disease hypertension and thyrotoxicosis it not uncommonly complicates constrictive pericarditis and atrial septal defect At times it is found in apparently healthy hearts (2)

Salient features of atrial fibrillation
1 absence of P waves which are replaced by irregular f' waves (or no sign at all of atrial activity)
2 normal QRS complexes irregular in time and sometimes varying in amplitude

REFERENCES

1 Fn elberg C D The e oj hageal electrocardiogram in the study of atrial activity and cardiac arrhythmias Am Heart J 19ɔ1 41 382
2 Evans W and Swann I Lone auricular fibrillation Brit Heart J 1954 16 189
3 Langendorf R Aberrant ventricular conduction Am Heart J 1951 41 700
4 Lown B et al Interrelationship of digitalis and potassium in auricular tachycardia with block Am Heart J 19ɔ3 45 589
5 Prinzmetal M et al *The Auricular Arrhythmias* Charles C Thomas Springfield Ill 19ɔ?
6 Simonson E and Berman R Differentiation between paroxysmal auricular tachycardia with partial A V block and auricularflutter Am Heart J 19ɔ1 42 387

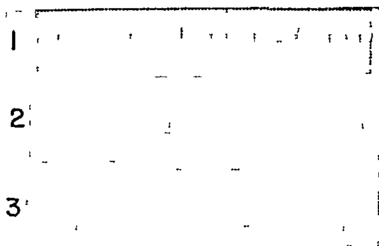


Fig 79 Atrial fibrillation with right axis deviation this combination is almost diagnostic of mitral stenosis

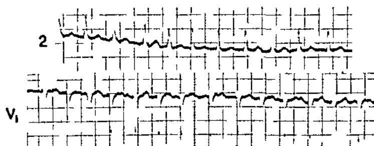


Fig 80 Atrial fibrillation with unusually regular ventricular response

The important causes of atrial fibrillation are rheumatic heart disease with mitral valve involvement coronary disease hypertension and thyrotoxicosis it not uncommonly complicates constrictive pericarditis and atrial septal defect At times it is found in apparently healthy hearts (2)

10

The Arrhythmias
A-V Nodal Arrhythmias

A-V NODAL PREMATURE BEATS AND TACHYCARDIA— A-V NODAL RHYTHM

When the impulse arises in the A-V node it travels up through the atria and down through the ventricle more or less simultaneously The impulse travels down to the ventricles by its normal paths and therefore the QRST sequence is normal but it is travelling in reverse through the atria and therefore the P wave is distorted (retrograde P wave) usually being inverted in several leads where it should be upright Three varieties of this pattern may occur

1 If the impulse reaches and spreads through the atria first the abnormal P wave closely precedes the QRS complex (fig 81a) the P-R interval being shorter than normal
2 If the impulse reaches the ventricles first the abnormal P wave is inscribed following the QRS (fig 81b)
3 If the impulse spreads through atria and ventricles simultaneously the P wave is lost in the QRS (fig 81c)



Fig 81 A-V nodal patterns a Abnormal retrograde P wave precedes QRS with short P-R interval (upper nodal rhythm) b Abnormal P wave follows QRS (lower nodal rhythm) c P wave coincides with QRS and is lost (mid nodal rhythm)

Review Tracings

Fig A

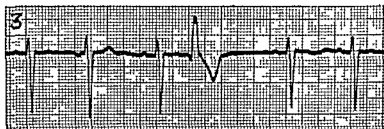


Fig B

For interpretations see page 213

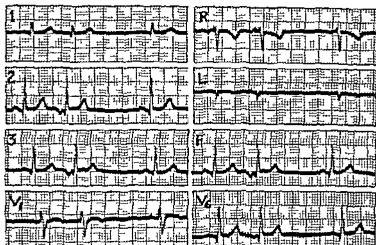


Fig 83 A V nodal premature beats Each lead contains a high nodal premature beat between two normal sinus beats P R interval of nodal beats is about 0 10 sec

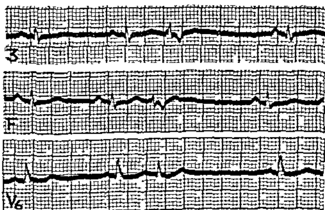


Fig 84 A V *nodal premature* **beats** The third beat in each lead is a low nodal premature beat—the P wave *follows* the QRS

Sometimes the A V node becomes exceedingly irritable and initiates a paroxysm of tachycardia **A V nodal tachycardia** (figs 85-87)

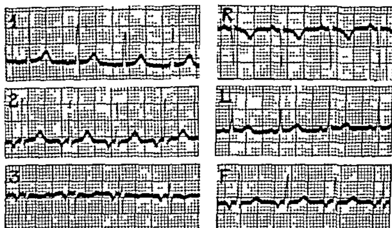


Fig 85 **A V nodal tachycardia** rate about 102 Note short P R interval with inverted P waves in 2 3 and aVF with upright P waves in aVR

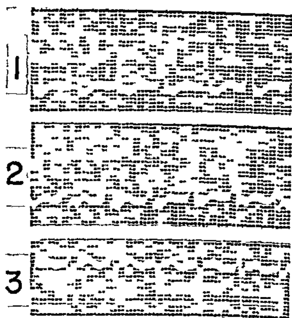


Fig 86 A V nodal tachycardia Inverted P waves following the QRS are clearly seen in lead 2

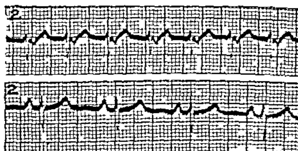


Fig 87 A V nodal tachycardia Two strips of lead 2 taken 10 minute apart upper strip shows nodal tachycardia with retrograde P waves lower strip shows normal sinus rhythm restored after 20 mg methoxamine intravenously

Sometimes when the S A node is suppressed by drugs or disease or is congenitally absent the A V node may come to the rescue and act as the pacemaker of the heart in its own intrinsic rhythm (25 to 60 beats per minute)—**A V nodal rhythm** (fig 88 89) All of these rhythms are relatively uncommon They are sometimes most easily recognized or confirmed by noting reversed polarity of P waves in aVR and aVF (fig 90)

The A V nodal premature beat with the preceding P wave can be distinguished from an atrial premature beat only by the shortened P R interval Similarly A V nodal tachycardia can only be distinguished from atrial tachycardia if the abnormal P wave is clearly visible accompanied by a shortened P R interval Although this distinction is generally accepted it is more arbitrary than accurate For an atrial ectopic focus adjacent to the A V node will obviously produce

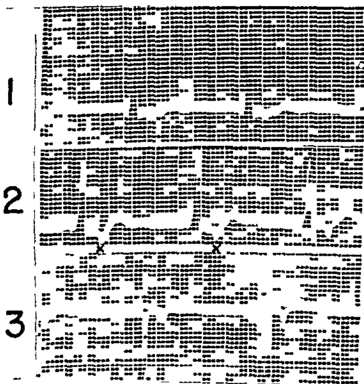


FIG 88 A V nodal rhythm From the same patient as figure 86 Ret

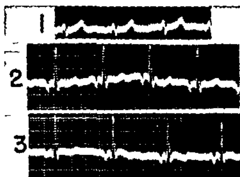


Fig 89 A V nodal rhythm Abnormal P waves closely precede normal QRS complexes in leads 2 and 3

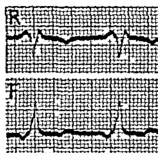


Fig 90 A V nodal rhythm Note that polarity of P waves in aVR and aVF is the reverse of normal i e upright in aVR and inverted in aVF

virtually the same P wave and P R interval as the A V node itself, and a focus adjacent to the S A node will produce as normal P waves as the S A node

And so again it is more logical to follow Prinzmetal s distinction between high (cephalic) low (caudal) and middle atrial foci by the P wave P R interval pattern if the P waves are inverted in leads 2, 3 and aVF and the P R interval is short the impulse originates in the caudal part of the atrium if the P waves are upright in 1 2 3 and aVF and the P R interval is normal or prolonged the focus of origin is in the cephalic region of one of the atria if the P waves are small or isoelectric in leads 1 2 3 and aVF the impulse originates in the central portion of the atrium

> Salient features of A V nodal rhythms
> 1 abnormal P waves closely preceding or following QRS or absent P waves
> 2 normal QRST sequence

CORONARY SINUS RHYTHMS

When P waves have the same shape and direction as those of A V nodal rhythm but are associated with a normal or prolonged (rather then short) P R interval there is reason to believe that such beats are likely to arise from the neighborhood of the coronary sinus Scherf (4) has applied the terms **coronary sinus rhythm** and **coronary sinus extrasystoles** to arrhythmias showing these features (fig 91)

WANDERING PACEMAKER

Rarely the site of impulse formation may vary between S A and A V nodes some beats arising from one and some from the other and others arising from the intermediate atrial muscle This phenomenon is known as **wandering or shifting pacemaker** (fig 92)

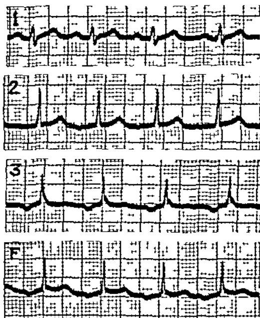


Fig 91 Probable coronary sinus rhythm Note inverted P waves in 2 3 and aVF with P R



Fig 92 **Wandering or shifting pacemaker** In lead 1 the first two P waves are inverted (A V nodal) while the last two are upright In lead 2 the first P wave is inverted (A V nodal) the next two are upright (S A) while the last is flat (mid atrial) In lead 3 the rhythm has settled down to regular A V nodal

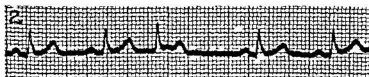


Fig 93 **Main stem extrasystole** The third beat is premature and has a normal shape A rhythmic sinus P wave is seen deforming the ST segment of the premature beat The pause following the premature beat is fully compensatory

MAIN STEM EXTRASYSTOLES

When a premature beat of supraventricular form is associated with no disturbance in shape or rhythm of P waves and is followed by a fully compensatory pause it is assumed that the beat arises in the main stem of the bundle of His (2) (fig 93) Such a beat thus has features of both ventricular and supraventricular premature beats—in form it is like a supraventricular beat but the undisturbed sequence of P waves and the compensatory pause are points in common with ventricular beats

✓ RECLASSIFICATION OF SUPRAVENTRICULAR ARRHYTHMIAS

In summary it may submitted that today the most rational classification of the supraventricular arrhythmias is as follows

Atrial

1 premature beats—usually recognizable as high middle or low in origin

2 paroxysmal tachycardia—sometimes (rarely) recognizable as high middle or low in origin—subdivided
   a without A V block (1 1)
   b with A V block (2–8 1)

3 fibrillation

A V nodal

1 premature beats—diagnosable if P wave is absent or follows QRS if P wave precedes indistinguishable from low atrial premature beat

2 tachycardia—diagnosable for certain only if P waves follow QRS if no P waves visible usually cannot be distinguished from atrial tachycardia if preceding P is visible cannot be distinguished from low atrial tachycardia

COMPARISON OF THE THREE MAIN TYPES OF PREMATURE BEATS

Let us consider for a moment the differentiation of the three main types of premature beat that we have now encountered—ventricular atrial and A V nodal In figure 94 seven possible variations on the premature beat theme are graphically represented The numbers of these seven beats are all circled The others are all sinus beats with a normal A V sequence

The first premature beat (3) is a premature ventricular beat followed by a compensatory pause (fig 51 A page 85) The second premature beat (5) is similar but is even more premature it is so premature that the next sinus beat finds the ventricle recovered and so initiates a normal ventricular contraction Such a premature beat shoved in between two normal beats with no compensatory pause is a true extra beat and is therefore an interpolated premature beat

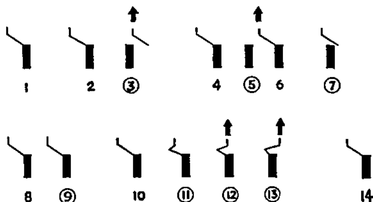


FIG 94 Diagram of atrio ventricular relationships in various forms of premature beats (see text)

(fig 51 B) The third premature beat (7) is barely premature, the impulse has already spread through the atria and the ventricular ectopic focus only just casts its vote in time (fig 50 B) The next premature beat (9) is atrial with normal A V sequence and is followed by an approximately normal cardiac cycle The next three premature beats (11 12 13) are A V nodal in 12 the impulse spreads through atria and ventricles simultaneously in 11 it traverses atria before ventricles (fig 83 page 117) and in 13 ventricles before atria (fig 84)

Ventricular contraction closes the A V valves Therefore in those premature beats where the ventricles contract *before* the atria i e beats 3 5 and 13 the subsequent atrial contraction vainly throws the blood against closed A V valves in such circumstances there is no where for the blood in the right atrium to go but back into the veins This may often be seen clearly in an accentuated venous a wave in the neck This phenomenon is termed a **cannon wave** Thus in some ventricular (3 and 5) and some A V nodal (12 and 13) beats cannon waves may be seen but they are *not* seen in atrial premature beats (9) where normal A V sequence is undisturbed

THE RELATIVE REGULARITY OF THE ARRHYTHMIAS

The most regular of the arrhythmias are the supraventricular tachycardias including atrial flutter with constant A V block

The most irregular is atrial fibrillation Between these extremes the other rapid arrhythmias dispose themselves They may be tabulated in decreasing order of their regularity as follows

atrial tachycardia
A V nodal tachycardia
sinus tachycardia
ventricular tachycardia
frequent premature beats
atrial tachycardia with varying A V block
atrial fibrillation

A knowledge of the relative degrees of regularity and irregularity is helpful but it should be borne in mind that this relationship is not invariable Atrial fibrillation for example may be surprisingly regular (fig 80 page 112) while atrial tachycardia may be surprisingly irregular

DIFFERENTIATION BETWEEN SUPRAVENTRICULAR AND VENTRICULAR TACHYCARDIAS

A *Clinical*

Two features of ventricular tachycardia help to distinguish it from supraventricular *asynchrony* between ventricles and *dissociation* between atria and ventricles Ventricular asynchrony leads to wide splitting of the heart sounds while A V dissociation produces cannon waves in the neck (whenever atrial contraction happens to fall just after ventricular) and variation in intensity of the first heart sound (5)

An uncomplicated supraventricular tachycardia should show none of these three findings but if a supraventricular tachycardia is associated with intraventricular block there should be signs of asynchrony without dissociation i e wide splitting of sounds without cannon waves or variation in first sound

Carotid sinus stimulation may help to differentiate in that ventricular tachycardia usually continues unaffected whereas a supraventricular tachycardia may be broken or at least temporarily slowed

These several points will be enough to distinguish many cases The

importance of these clinical observations should be emphasized because the standard electrocardiogram is often inadequate for separating supraventricular tachycardia with intraventricular block from ventricular tachycardia

B *Electrocardiographic*

The distinction between typical ventricular and supraventricular tachycardias is easy Difficulty arises in separating ventricular tachycardia from a supraventricular tachycardia combined with intraventricular block and the crux of such separation lies in identifying the P waves If these are identifiable their constant or varying relationship to the QRS will respectively indicate supraventricular or ventricular tachycardia If they are not recognizable even in V1 more specialized leads may be informative a precordial lead taken from an interspace above V1 (atrial lead point see fig 2 page 2) may reveal P waves and for this the CR connection may be more useful than the V If this is unsuccessful a lead known as S5 may be tried (3) for this the right arm electrode is placed over the manubrium and the left arm electrode in the 5th right interspace close to the sternal border with the switch set for standard lead 1 If this fails an esophageal lead will almost always be successful in displaying P waves Alternatively if a tracing is taken during the administration of pronestyl intravenously the ventricular rate will often be seen to slow under its influence and P waves will become apparent in the now lengthened intervals between ventricular complexes (1)

In summary

Clinically
- (a) look for
  - 1) wide splitting of heart sounds
  - 2) variation in intensity of 1st sound
  - 3) cannon waves
- (b) observe effect of carotid sinus stimulation

In the electrocardiogram *cherchez le P*
- 1) in standard tracing especially in V1
- 2) in lead from interspace above V1,
- 3) in lead S5
- 4) in esophageal lead
- 5) during intravenous pronestyl administration

REFERENCES

1 Bernstein I M et al Intravenous procaine amide as an aid to differentiate auricular flutter with bundle branch block from paroxysmal ventricular tachycardia Am Heart J 1954 48 82
2 Fletcher E Extrasystoles arising from the main bundle of His Brit Heart J 1955 17 566
3 Lian Casimatis and Hebert Interet de la derivation precordiale auriculaire S dans le diagnostic des troubles du rythme auriculaire Arch Mal Coeur 1952 45 481
4 Scherf D and Harris R Coronary sinus rhythm Am Heart J 1946 32 443
5 Schrire V and Vogelpoel L The clinical and electrocardiographic differentiation of supraventricular and ventricular tachycardias with regular rhythm Am Heart J 1955 49 162

Review Tracing

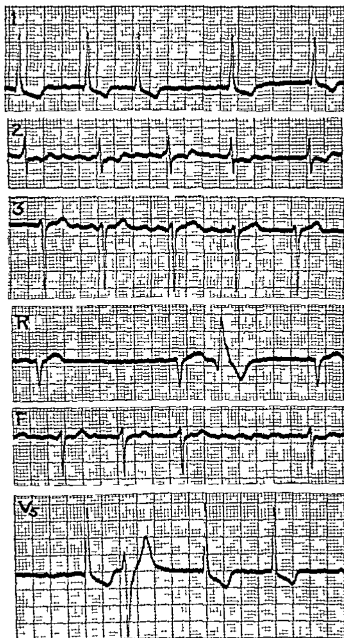


Fig C For interpretation see p 213

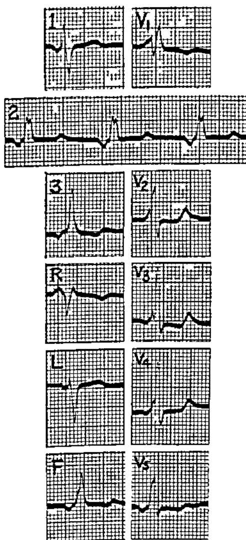


FIG D For interpretation see p 213

11

Intra-Atrial, Sinu-Atrial and Atrio-Ventricular Block, A-V Dissociation

INTRA ATRIAL BLOCK

If the impulse takes longer than normal to activate the atria, i.e., if the P wave is widened, intra atrial block is said to be present. The upper normal limit of P wave duration is not universally agreed upon but the most satisfactory limit is probably 0.11 second. The criterion therefore for diagnosing intra atrial block is a P wave with a duration of 0.12 sec. or more (fig. 95). Further evidence of block is to be found in deep notching of the P wave with a distance between peaks ("peak interval") of more than 0.04 sec. (fig. 95 C).

Intra atrial block is not uncommon and is most often seen in coronary disease, mitral disease and in association with left ventricular hypertrophy (1). It probably often represents atrial enlargement rather than true block and is thus analogous to the term intraventricular block applied to the QRS interval of 0.11 sec. or more when this is due to ventricular enlargement (see page 73).

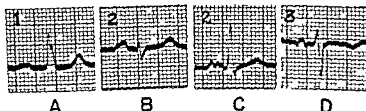


FIG. 95. Intra atrial block. Four samples from four different patients. A and B show widening of P wave without significant notching. C and D show marked notching with wide peak interval (more than 0.04 sec.).

When the heart actually drops a beat it means either that the S A node has failed to initiate the impulse (S A block) or much more likely that the impulse after traversing the atria has been unable to get through the A V conducting tissues (A V block) Both these types of block may be recognized in any lead in which P waves are clearly formed

S A BLOCK

S A block is divided into partial and complete

a **Partial S A block** consists in the more or less infrequent suppression of impulse formation at the S A node with the result that occasional beats are completely dropped This is recognized in the tracing by the occasional absence of the entire P QRS T sequence (fig 96)

b **Complete S A block** exists when *no* impulses are formed in the S A node—the S A pacemaker is completely knocked out This situation is also referred to as **atrial paralysis** or **atrial standstill** In these circumstances one of two things can happen either 1) a lower pacemaker usually the A V node comes to the rescue and takes over the job of pacemaking or 2) cardiac standstill continues and the patient dies If 1) occurs and the ventricles proceed to beat independently **nodal or ventricular escape** is said to have occurred and the heart s rhythm is called idioventricular Complete S A block is thus one of the two mechanisms (much the less common) leading to the development of **idioventricular rhythm**

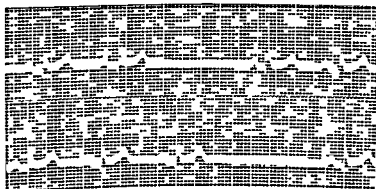


Fig 96 **Partial S A block** In each of the two strips a complete P QRS T sequence has been dropped

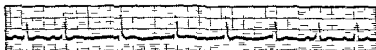


FIG 97 Transient S A block induced by a deep breath After the second beat P waves are suppressed and A V nodal escape occurs for the next four beats The S A node then resumes control for the last two beats

Complete S A block is recognized in the electrocardiogram by the complete absence of the entire P QRS T sequence i e by a completely straight and unadorned baseline this continues until if the patient is fortunate idioventricular QRST complexes appear at a slow rate (usually 30 to 40) while P waves remain absent

S A block is rather rare but it can be produced by a wide variety of causes *drugs* such as digitalis quinidine and salicylates *diseases* such as coronary disease and acute infections *physiologic* disturbances such as carotid sinus sensitivity and increased vagal tone in susceptible patients even a deep breath may suppress the S A node long enough for nodal escape to occur (fig 97)

> Salient features of S A block
> a Partial occasional absence of P QRS T sequence
> b Complete
> 1 P waves absent
> 2 QRST sequence at idioventricular rate (30–40)
> 3 QRS interval normal or prolonged depending on site of ventricular pacemaker

A V BLOCK

A V block is also divided into partial and complete

a **Partial A V block** covers all defects of A V conduction short of complete dissociation between atria and ventricles In mild cases the impulse merely takes longer to travel from atrium to ventricle it is delayed in the A V conducting tissues and A V conduction time (P R interval) is prolonged In severer grades the atrial impulse is at times held up completely at the A V node or bundle fails altogether to reach the ventricles and a dropped beat results Such dropped beats are recognized in the tracing by the presence of isolated P waves not followed by ventricular complexes

Partial A V block is conveniently divided into degrees sub divisions vary with different authors some dividing it into two and others into three degrees A convenient classification is as follows

1st degree partial A V block—simple prolongation of the P R interval without dropped beats (fig 98)

2nd degree partial A V block—beats are dropped more or less frequently up to a maximum of every third beat i e a 3 2 ratio (fig 99)

3rd degree partial A V block (high grade A V block)— at least every other beat is dropped i e a 2 or more than 2 to 1 A V block (fig 100 A and B) In atrial flutter a 7 or 8 to 1 ratio is sometimes seen

Second and third degree partial A V blocks require amplification Dropped beats can occur against three differing backgrounds 1) most commonly the P R interval is prolonged in tho e cycles where the beat is not dropped i e both 1st and 2nd or 3rd degree block are present (fig 100 A) 2) uncommonly the P R interval may be normal i e there is 2nd or 3rd degree block but no 1st degree block (fig 100 B) or 3) the P R interval may progressively lengthen until a beat is dropped This is known as **Wenckebach s phenomenon** (figs 100 C and 99) The P R interval may begin within normal limits or it may be somewhat prolonged then with each successive beat the P R interval gradually lengthens until finally an impul e fails to reach the ventricles and a beat is dropped Following the dropped beat the P R interval reverts to normal or near normal and the sequence is repeated

It is worth drawing attention to two clinical findings that characterize this phenomenon

1 In listening to the heart one hears it slowing down (for as each

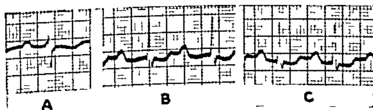


Fig 98 **1st degree partial A V block** The P R interval is prolonged A P R interval is 0 ?6 seconds B P R interval is 0 34 seconds and in this case is caused by digitalis C Same patient as B showing effect of forced inspiration note increase in rate and decrease in P R interval to 0 2? seconds

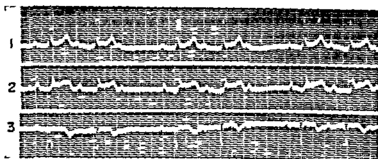


Fig 99 2nd degree partial A V block Every third ventricular beat is dropped This tracing also shows the Wenckebach phenomenon the first P R interval is 0 24 seconds the second is 0 3[illegible] seconds the third beat is blocked and the cycle begins again

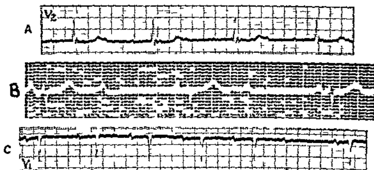


Fig 100 Partial A V block A 3rd degree partial block (3 to 1) with prolonged P P interval of 0 36–0 40 seconds

B 3rd degree partial block (2 to 1) with normal P P interval of 0 15 seconds

C Wenckebach s phenomenon The P R intervals are successively 0 24 0 29 0 33 0 34 and 0 36 seconds and then a beat is dropped the P R interval then returns to 0 24 seconds

successive P R interval lengthens the total R R interval lengthens with it proportionately) then as the beat is dropped there is a pause following the pause the heart accelerates for it now has a shorter near normal P R interval

2 If the Wenckebach phenomenon occur in a heart with the presystolic murmur of mitral stenosis the murmur gets left further and further behind in diastole as A V conduction time lengthens until what was the late diastolic murmur has become mid-diastolic

then comes the pause and with the first beat following it the murmur returns to its presystolic phase This finding was long ago correlated by Mackenzie, it affords indirect confirmation of the belief that atrial contraction is largely responsible for the presystolic accentuation of the murmur of *mitral stenosis*

b **Complete A V block** When *no* impulses pass the A V barrier complete A V block sometimes called **complete A V dissociation** has developed Bilateral bundle branch block rather than blockade at the A V node or in the main bundle is sometimes the cause of complete A V block (3) As with the less common complete S A block two possibilities now exist Either the ventricles remain inactive (***ventricular standstill***) and the patient dies or more likely the A V node (or a lower pacemaker) takes over and controls the ventricles (**nodal or ventricular escape**) In this event the atria continue to beat in their own time and the ventricles now beat in a slower tempo **idioventricular rhythm** For example the atria may continue to beat at a sinus rate of 80 while the ventricles perform at 30 (fig 101) This independence is readily recognized in the tracing by the lack of relationship between the slow ventricular complexes

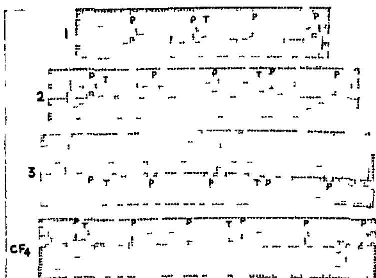


Fig 101 **Complete A V block** P waves and QRS complexes are entirely independent Atrial rate 80 ventricular rate 31 Inverted atrial T waves (Ta or T_P waves) are plainly visible shortly following P waves in leads 1 and CF4

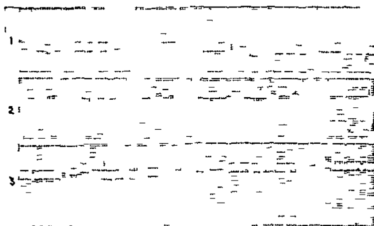


FIG 102 **Complete A V block** P waves and QRS complexes are independent The rhythm is complicated by the occurrence of **premature ventricular beats** As the QRS complexes are prolonged and show left axis deviation the idioventricular pacemaker is presumably an ectopic focus situated in the right ventricular myocardium

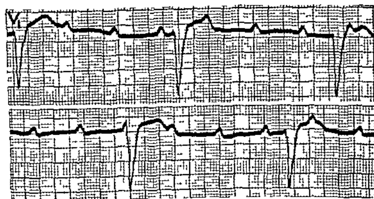


FIG 103 **Complete A V block** The two strips form a continuous record P waves and QRS complexes are independent at ventricular rate of 28 and atrial of 96

and the more frequent P waves Each maintains its own rhythm without regard for the other

If the idioventricular rhythm is initiated by the A V node or nearby junctional tissues the QRS interval and complex will be normal if the rescuing pacemaker is in the ventricular muscle itself then the QRST cycle is bizarre with prolonged QRS interval and in form resembles a premature ventricular beat (figs 102 and 103)

In case there should be any doubt it is perhaps worth clarifying the difference between idioventricular and A V nodal rhythm We have seen that the A V node can control the ventricles in both these rhythms Idioventricular rhythm however implies that the ventricles have their own pacemaker all to themselves and are beating independently of the atria the atria are either beating in their own time (complete A V block) or they are inactive (complete S A block) A V nodal rhythm on the other hand means that *both* the ventricles *and* the atria are under the control of the A V node—the A V node is the pacemaker of the whole heart

The transient ventricular standstill that occurs when block becomes complete is the underlying basis for true Adams Stokes attacks Complete A V block is an easy bedside diagnosis Suspicion is immediately aroused by the slow ventricular rate Then signs of dissociation between atria and ventricles (variation in intensity of first heart sound with an occasional explosive first sound— bruit de canon independent a waves and cannon waves in the jugulars) clinch the diagnosis Complete block may sometimes be distinguished from 2 1 A V block by exercise for this tends to double the rate in 2 1 block but has no effect on the idioventricular rhythm and rate of complete block

A V block is common It is most often produced by coronary and rheumatic disease varying in incidence with the age group Less frequently congenital heart disease syphilis diphtheria or uremia produces it Digitalis and quinidine in toxic dosage also often induce this type of block

Salient features of A V block

a Partial—impaired A V conduction indicated by
   1 prolonged P R interval (1st degree)
   2 dropped beats—P waves not followed by QRST (2nd and 3rd degrees)

b Complete—atria and ventricles independent
   1 P waves at sinus rate (60–90)
   2 QRST at idioventricular rate (30–40)
   3 QRS interval normal or prolonged depending on site of ventricular pacemaker

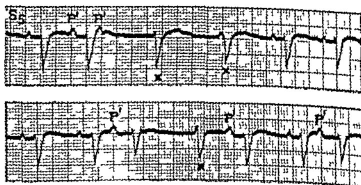


FIG 104 Nodal es pe The tw o trips are a continuous record of lead R_4 Dominant rhythm A i l everal **atrial premature beats (P')** Following two of these pr t le t longer than usual R P intervals are terminated by nodal p l

NODAL AND VE [illegible] ESCAPE

Escape has be [illegible] ioned above in connection with both S A and complete A V b [illegible] be regarded as a safety mechanism whereby the he [illegible] when the higher pacemaker fails Usually the escaping p [illegible] the A V node itself (**nodal escape**), but if this also det [illegible] he ventricular myocardium may take over (**ventricular e** [illegible] ape may occur for a single beat terminating an unu [illegible] pau e or for but a few beats or may usher in a more [illegible] idioventricular rhythm The escape beat is recognized [illegible] ence of a preceding P wave and 2) its *late* appearance [illegible] nly after the next expected beat has failed to appear [illegible] ded by a longer interval than the previous R R interval [illegible] 104 and 97) The QRS complexes of nodal escape beat [illegible] ntricular form whereas ventricular escape beats have th [illegible] rm of ectopic ventricular beats

MARKED BRADYCARDIA

As complete A V [illegible] the commonest cause of marked bradycardia it may [illegible] f place to call attention to other causes A rate of 30 to 50 m [illegible]

1 idioventricular rhythm re ulting from complete A V or S-A block (figs 101 103)
2 high grade partial A V block with a inus rate of say 80 and a ventricular rate of 40 (2 1 block) (fig 100 A and B page 135)

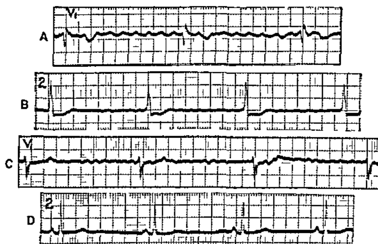


Fig 105 Various bradycardias A Atrial flutter with high grade (5 to 1) A V block and ventricular rate of 31 B and C Atrial fibrillation with complete A V block note that ventricular rhythm is absolutely regular in B at rate 38 in C at 32 D Sinus bradycardia at rate 40–45

3 A V nodal rhythm
4 atrial tachycardia (flutter) with high grades of A V block e g 8 1 block with an atrial rate of 320 giving a ventricular rate of 40 (fig 105 A)
5 atrial fibrillation with marked A V block (fig 105 B and C)
6 sinus bradycardia (fig 105 D)

✓ PROLONGED QRS INTERVAL

We can now summarize the conditions associated with a prolonged intraventricular conduction time A prolonged QRS interval occurs

a When the impulse is initiated by an ectopic ventricular focus
  1 premature ventricular beat
  2 ventricular tachycardia
  3 idioventricular rhythm (from a low pacemaker)

b When intraventricular conduction is slowed
  1 intraventricular block
  2 ventricular aberration

c When conduction to one ventricle is accelerated
  1 Wolff Parkinson White syndrome

A V DISSOCIATION

It has been mentioned that complete A V block is sometimes referred to as complete A V dissociation and it is certainly reasonable that any rhythm characterized by independent action of atria and ventricles should be so called However the type of independent rhythm that is usually meant when the term A V dissociation is used without qualification has nothing to do with A V block

If the excitability of the S A node becomes unduly depressed or if the A V node becomes unduly excitable we have seen that the A V node may usurp control of the heart producing A V nodal rhythm In this rhythm the impulse travels backwards into the atria from the A V node and forward into the ventricles more or less simultaneously If however backward conduction into the atria is blocked (retrograde block) the atria are protected from A V nodal control and continue to obey the S A node When this occurs the rhythm is properly called A V dissociation For this disturbance to develop there are thus two fundamental requisites 1) the ventricular pacemaker must be firing somewhat faster than the S A node and 2) retrograde block must be present Forward conduction through the A V tissues can still take place however so that when the sinus impulse happens to land at a strategic point in the cardiac cycle (when junctional tissues and the ventricles are not refractory) it is conducted to the ventricles This occasional conducted beat interferes with the rhythm of the otherwise independently beating ventricles The phenomenon is known as interference and the conducted beat as an interference beat

The terminology of this rather difficult subject has been unnecessarily confused by the experts The use of the term dissociation for two fundamentally different mechanisms as mentioned above is the first point of confusion The second is in the application of the term interference As used here it refers to the fact that one pacemaker interferes with the rhythm of a second pacemaker This was the sense

implied by Mobitz when he originally introduced the term **interference dissociation** and by Scherf when he later modified the term to **dissociation with interference** Other high authorities (2) ignore this use of the term and employ it in an entirely different sense—a sense no less correct in the terminology of physics but nonetheless unfortunate because of the confusion thereby encouraged in an already difficult subject Further because of this difference of meaning it is necessary for this school to retain an additional term ventricular capture for the phenomenon of the isolated conducted sinus beat

In the electrocardiogram the P waves are seen to overtake and pass the QRS complexes (fig 106) When the P wave falls sufficiently far beyond the QRS the impulse will be conducted to the ventricles making them beat prematurely and thus interfering with their otherwise independent rhythm (x in fig 106) Thus a series of dissociated beats is punctuated by a normally conducted beat

A V dissociation may be caused by factors which depress the S A node such as digitalis or by abnormal conditions that make the A V node more excitable such as rheumatic fever and acute infections It not uncommonly complicates the course of coronary disease

REFERENCES

1 Bradley S M and Marriott H J L Intra atrial block Circulation 1956 14 1073
2 Katz L N and Pick A *Clinical Electrocardiography* The arrhythmias Lea and Febiger Philadelphia 1956
3 Rosenbaum M B and Lepeschkin E Bilateral bundle branch block Am Heart J 1955 50 38

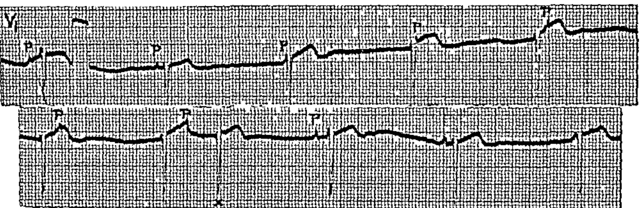


Fig 100 A V dissociation with interference The two strips are a continuous record Ventricles are beating slightly faster (42) than atria (40) The P wave appears first in front of the QRS but gradually overtakes it and by the last beat of the first strip has emerged well beyond the QRS to deform the ST segment When the P wave falls sufficiently far beyond the QRS (second P in lower strip) the impulse is conducted to the ventricles (third QRS in lower strip) Thus the atria interfere with the otherwise independent ventricular rhythm

Review Tracings

Fig E



Fig F

For interpretations see page 213

12

Myocardial Infarction

EXPERIMENTAL CONSIDERATIONS

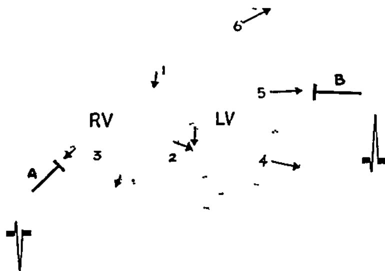
If a branch of a dog's coronary is tied and an electrode is placed on an area of myocardium supplied by the occluded vessel the T waves in the derived tracing soon become inverted If the ligature is then removed and the flow of blood to the muscle reestablished the inverted T waves soon return to normal The T wave inversion is therefore clearly the result of simple ischemia Inverted T waves form the basis of the **pattern of ischemia** in the clinical tracing

If when T inversion occurs the ligature is allowed to remain in place a dramatic change in the pattern shortly develops within a minute or two the ST segment becomes strikingly elevated dragging up with it and obliterating the inverted T wave If at this stage the tie is removed the tracing gradually passing back through the inverted T stage again reverts to normal ST elevation representing a stage beyond ischemia but still reversible is known as the **pattern of injury**

If when the pattern of injury is fully developed the tie is left in place a further striking change eventually occurs The entire QRS complex becomes inverted to produce a QS complex while the ST segment comes back to the isoelectric line and the T wave once more assumes its normal upright contour If this pattern is allowed to persist for long before the ligature is removed it is found to be irreversible—no matter how long and patiently you wait a QS pattern will continue to be recorded from the damaged area Irreversible structural

changes have occurred and the new pattern is called the **pattern of necrosis**

It is not at first obvious why necrosis should produce this pattern If a tracing is taken with an electrode *inside* the cavity of the left ventricle however an identical QS pattern is recorded It is easy to rationalize this A glance at figure 26 (repeated below) will remind us that all the impulses (dipoles) are travelling centrifugally from the ventricular cavity—they are all speeding outwards through its walls with their negative tails towards the cavity The deflection recorded from inside the ventricle will therefore be entirely negative that is QS in form Now when a patch of the ventricular wall is dead and an electrode is placed over the necrotic patch it is as though the electrode outside is looking through a window into the cavity and reproducing what it sees within Expressed differently the dead muscle can no longer actively transmit impulses it now acts like a passive conductor of what is beyond it i e intracavitary negativity



This 'window' theory has held sway and served its purpose for many years It is probably more accurate however, to think of the development of Q waves in a somewhat different light If a segment of myocardium is knocked out, electromotive forces cease to traverse it There is thus a loss of forces directed towards the electrode placed over the inert muscle and this results in a negative deflection (Q wave) By the same token there will be a relative gain in forces directed away from the inert area and this may be indicated by increase in the size of the positive deflection in leads taken from the opposite surface of the heart This concept has been helpful in explaining some of the less classical patterns of infarction encountered several of which will be discussed later in this chapter

CLINICAL INFARCTION

These three changes observed in the experimental heart—T wave inversion ST elevation and the appearance of Q waves—form the basis of infarction patterns as we see them clinically Around any patch of infarcted muscle there is a less damaged zone which will produce the pattern of injury and outside this an even less affected area from which the pattern of ischemia will be derived In the experimental heart these zones can be picked up individually with the use of small electrodes placed directly on the epicardium (**direct leads**) Clinically the nearest one can get is two or three inches from the myocardium on the outside of the chest (**semi direct leads**) A natural result of this is that the precordial pattern is usually a composite picture combining the patterns of ischemia injury and necrosis all in one QRST sequence—the relatively distant electrode is influenced by all three zones instead of only one

The first step then in diagnosing infarction from the electrocardiogram is to know what changes to look for Those changes are 1) the **fresh appearance of Q waves or the increased prominence of pre existing ones** 2) **ST segment elevations** and 3) **T wave inversions** Only Q wave changes are absolutely diagnostic of infarction (necrosis) but changes in the ST segments and T waves may be suspicious and provide strong presumptive evidence The Q ST and T changes all have special characteristics which should be thoroughly appreciated The Q wave is often wide as well as deep any Q measuring 0 03 seconds or more in width (from onset to nadir) is highly suspicious of infarction The deviated ST segments typically show an

upward convexity The fully developed T waves are pointed and consist of two symmetrical limbs well likened to an arrow head These three changes are summarized in figure 107 Note that these changes are registered in **leads which face the area of damage,** and it is convenient to refer to them collectively as "indicative" changes

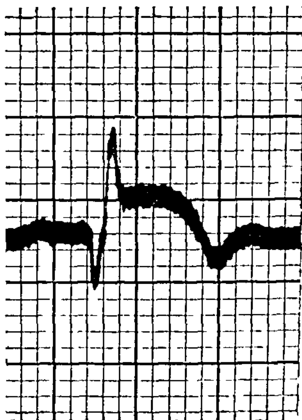


Fig 107 Acute myocardial infarction The three indicative changes—Q wave ST elevation T inversion

The two main types of infarction u ed to be called anterior and posterior But becau e the term posterior is anatomically inac curate as it was applied to the surface of the heart resting on the diaphragm it is becoming more common and certainly better practice to use the terms inferior or diaphragmatic when referring to lesions of this wall

If you hold a heart in your hand it is at once obvious that there are no clear cut surfaces or boundaries any walls defined are at best rough approximations The four walls usually referred to in dis cussions of infarction are anterior lateral inferior and true posterior (to distinguish it from the false posterior of the older terminology) These will be adopted here

It was stated above that indicative changes develop in leads *facing* the area of damage Opposite or reciprocal changes (i e no Q wave with perhaps some increase in height of R wave depressed ST segments and tall upright T waves) meanwhile appear in leads facing

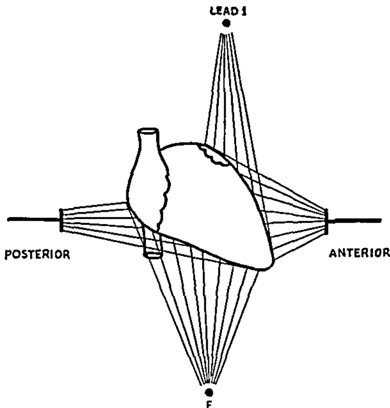


FIG 108 Illustrating how the anterior chest leads and lead 1 both face the same anterior (really antero superior) surface of the heart while the posterior chest leads and the lower pole (F) of lead 3 face the inferior (diaphragmatic) surface

the diametrically opposed surface of the heart Fortified with a little imagination and with a glance at figure 108 it is now relatively simple to decide what changes will occur in which leads when various surfaces of the heart are involved The only lead which exclusively faces the inferior surface is aVF and this is therefore the most important lead in the diagnosis of inferior infarction in some cases it is the first or only lead to show indicative changes but leads 2 and 3 each being partly composed of aVF, usually show these changes also Reciprocal changes are usually seen in leads 1 aVL and some of the precordial leads In anterior infarction indicative changes occur in precordial leads and in leads 1 and aVL while reciprocal

changes develop in leads that face the infero-posterior surface (2, 3 and aVF) In **lateral** wall infarction leads aVL and V_4 and V_6 are most likely to show indicative changes and reciprocal changes may sometimes develop in leads taken furthest to the right (V_1, V_{3R}, etc) None of the routine 12 leads faces the **true posterior** surface of the heart and so infarction of this wall must be inferred from reciprocal changes occurring in precordial leads especially V_1 and V_2

The limb lead patterns associated with the two main types of infarction anterior and inferior are so well engrained in many minds that it is perhaps worth summarizing them separately Anterior infarction produces indicative changes in lead 1 and for this reason has been called Q_1T_1 type infarction Inferior infarction produces indicative changes in lead 3 and has therefore been called Q_3T_3 type infarction In anterior infarction indicative changes are also frequently seen in aVL and reciprocal changes appear in 3 and aVF In inferior infarction indicative changes are also found in 2 and aVF while reciprocal changes develop in 1 and aVL These changes are summarized in figure 100

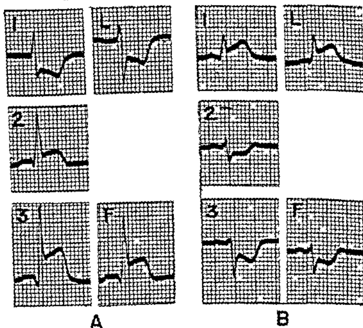


FIG 100 Characteristic early changes of acute infarction in the limb leads A **Inferior infarction** B **Anterior infarction**

It is important to realize however that the characteristic changes of infarction may appear *only* in the precordial leads the limb leads remaining normal or near normal

Evaluation of Q_3

As a prominent Q wave in lead 3 is one of the hallmarks of inferior infarction but is also sometimes a perfectly normal finding its evaluation is often difficult (9) It is more likely to be abnormal if it is wide (more than 0 03 sec) if it is associated with Q waves also in 2 and aVF and if it is followed by a slurred upstroke into the R wave

A simple test is sometimes helpful deep inspiration will usually cause an innocuous (positional) Q_3 to disappear or materially decrease (fig 110) whereas the Q_3 of infarction is relatively unaffected by this simple maneuver (4) (fig 111)

At times but by no means invariably it is helpful to refer the decision to the aV leads (29) for lead 3 connecting left arm and left leg represents the difference between aVF and aVL (3 = aVF − aVL) If the initial deflection of the QRS in aVF is less positive (or more negative) than the corresponding deflection in aVL there will be an initial negative (Q) wave in 3 And so if there is no Q wave in either aVL or aVF but the R wave in aVF is not so tall as the R in aVL (fig 112 A) there will be a Q wave in 3 This will clearly not be a pathological Q wave as it results simply from difference in the height of normal R waves In such a situation the aV leads give an immediate

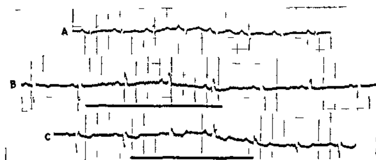


Fig 110 The three strips are lead 3 from three different patients A Variation in QRS amplitude and direction with normal respiration B The effect of forced deep inspiration on a deep Q_3 Duration of inspiration indicated by black marker Note complete disappearance of Q wave at height of inspiration C A similar illustration showing the effect of an uninvited sigh on a deep Q_3

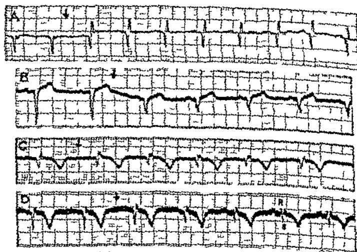


FIG 111 Effect of deep inspiration on Q_3 in four patients with inferior infarction Inspiration began at the arrow in each strip Note that there is relatively little effect on the negative wave (compared with normals in fig 110) In D the Q wave is replaced by a small initial R wave but an appreciable negative (S) wave persists

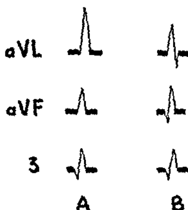


FIG 112

favorable answer If on the other hand there is a Q wave in aVF (fig 112 B) one has merely transferred the burden of proof to aVF and it then has to be decided whether the Q in aVF is of abnormal significance or not

Further observations

Some important general points must be borne in mind

a Time relationships are of great importance Rarely, no changes develop in the tracing for several days or even for two or three weeks Usually however they begin to make their appearance within the first few hours ST segment changes appear early and progress At this

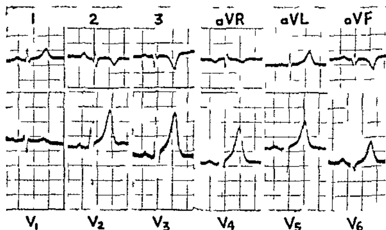


FIG 113 Acute inferior myocardial infarction Note indicative changes in 2 3 and aVF with reciprocally tall pointed T waves in precordial leads

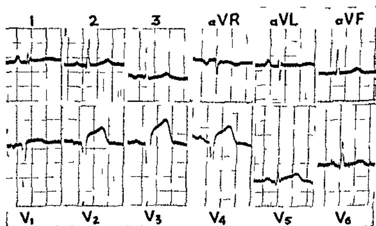


FIG 114 Acute anterior myocardial infarction Note standard leads do not show diagnostic changes while characteristic Q and ST deviations are conspicuous in V_1 to V

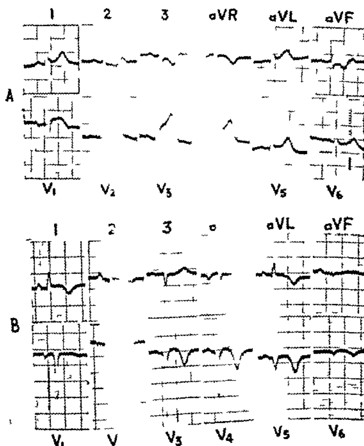


Fig 115 A Acute tero lateral myocardial inf rction Note early changes of ant f rcti n include t ll hal T waves which may be mistak th lat r tall T waves f o infarction B Tracing taken s It shows th full ped pattern of extensive anterior n r te lateral st ost ted by the Q and T changes evi r to th left as V and V ly changes are present in aVL

stage the T waves later to become inverted actually become taller appearing as an upw d extension of the rising ST segment (23) This early tall T wave may be mistaken for the later tall T wave of reciprocal leads and an early anterior infarction may thus be wrongly labelled as inferior and vice versa (fig 115 A and 116 A) sometimes

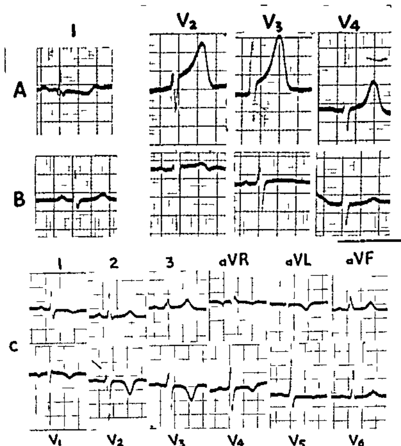


FIG 116 A **Acute antero septal myocardial infarction** Note the tall T waves in V_{2-4} where they will later be deeply inverted

B Taken just a few hours later note striking changes in T waves tracing is now remarkably normal

C Taken six days later shows fully developed pattern of antero septal infarction Note that no pathological Q waves have developed but the infarction can be diagnosed with certainty from the striking evolution in the ST T pattern

this tall T wave is associated with striking depression of the ST segment (25) and then of course the reciprocal pattern of an inferior infarction is even more closely simulated (fig 117) To add to the confusion similar tall T waves are occasionally seen as an early stage of inferior infarction (8) in such cases this may well represent a premonitory stage of diaphragmatic wall ischemia before actual infarction has occurred Similar but persistent tall T waves are a not uncommon finding in patients with angina (27)

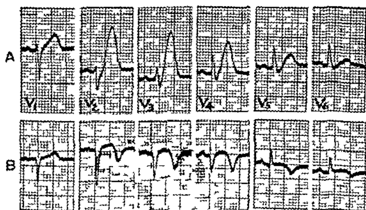


Fig 11 Acute anterior infarction Note unusual early stage in A with tall T waves and *depressed* ST take off B taken a few days later shows typical evolution of anterior infarction

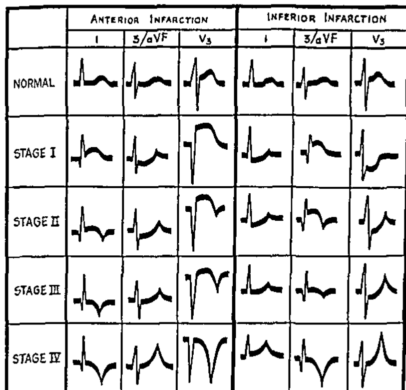


FIG 118 **Acute myocardial infarction** Stages of evolution in the patterns of anterior and inferior infarctions

Q waves may appear early or may not develop for several days Whenever the various changes appear they tend to evolve in a fairly typical sequence (fig 118) In the indicative leads the ST segments rise higher and higher and then begin to return to the baseline while the T waves develop progressively deeper inversion finally after weeks or months the T waves may become shallower and finally return to normal Thus ST changes are usually the most transitory the T changes are more lasting but the Q waves are the most likely to remain as a permanent record of the myocardial scar Persistent ST segment changes suggest the possibility of ventricular aneurysm (26)

The model sequence of changes depicted diagrammatically in figure 118 is not invariable but there is always some evolution of the pattern along similar lines and to diagnose an *acute* infarction such evolution must be in evidence For there is no means of being certain in a single tracing whether the typical changes of infarction are due to an acute process or are the remnants of an old one ST segment deviations are least likely to represent an old process but even they may sometimes endure for a long time and rarely are permanent Progressive changes from day to day are the conclusive evidence of an active acute process

b The electrocardiogram should be considered *confirmatory* of the clinical impression and should not supersede it If the patient is suspected clinically of having sustained a myocardial infarct he should emphatically be treated as such even if his tracing is completely normal The looked for changes may be late to appear or rarely they may never appear in the routine leads although the infarction is a clinical certainty For factors favoring missed diagnoses of infarction see page 169

c A T wave pattern of some importance is the **T_1 lower than T_3** (or aVF) pattern T_1 is often found normally lower than T_3 (or aVF) in vertical hearts with right axis deviation It is also abnormally present in early left ventricular strain before frank inversion of T_1 has occurred If both vertical heart and left ventricular strain can be excluded such a pattern is extremely suspicious of an anterior infarction either old or recent (24)

d Apart from the changes specific for acute infarction other abnormalities frequently appear The tracing often shows low voltage and the QT duration is frequently prolonged reaching its maximum in the second week Any arrhythmia or block may develop notably atrial fibrillation ventricular premature beats tachycardia or fibrillation A V block or bundle branch block

Salient features of acute myocardial infarction

	Anterior	*Inferior*
1 Indicative changes (Q ST elevation T inversion) in leads	1 aVL anterior chest	3 aVF posterior chest
2 Reciprocal changes in leads	3 aVF posterior chest	1 aVL anterior chest
3 Progressive changes in pattern from day to day		

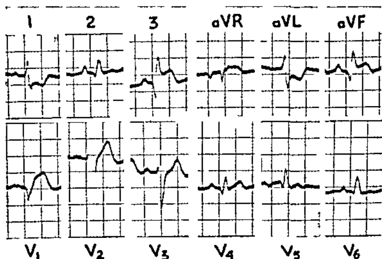
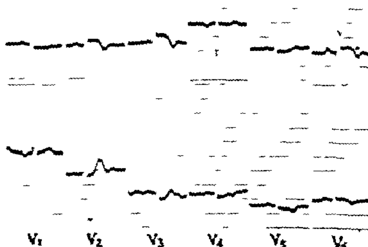


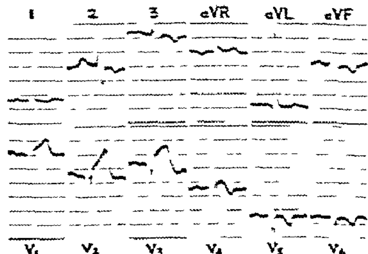
Fig 119 Acute inferior myocardial infarction Note indicative changes in aVF 2 and 3

Complex patterns

Frequently the pattern observed is not so pure as the ones so far described If the anterior and inferior walls of the left ventricle are both involved in the process **antero inferior infarction,** varying combinations of the changes typical of each may occur (3) Sometimes an inferior infarction develops in a heart that has suffered a



F[illegible] 12 Acute infero-lateral infarction [illegible] The ST T [illegible]



F[illegible] Acute anterior infarction [illegible] left [illegible] hypertrophy and strain [illegible] What [illegible]

previous anterior infarction or vice versa in such circumstances the current infarction producing changes opposite (or reciprocal) to the changes of the previous infarction may tend to "normalize" the tracing so that it looks "better" than it did before the second occlusion

Bundle branch block producing as it does bizarre QRS ST and T changes of its own may completely mask the changes of a superimposed infarction One of the most difficult diagnoses to make is that of infarction in the presence of left bundle branch block (11) (fig 122) If a previous tracing is available showing the uncomplicated block pattern then the appearance of fresh Q waves (especially over the left ventricle where they are not found in uncomplicated left bundle branch block) or a decrease in amplitude of R waves over the left ventricle (13) or a change in the elevation or shape of ST segments or T waves may provide a suspicious clue but certainty can only be expressed if definite evolution in a pattern suspicious of infarction is noted from day to day

Right bundle branch block is less likely to cause confusion (12) Both anterior and inferior infarction patterns can often be seen clearly superimposed on the block pattern (fig 123 and 124) It should be remembered that the block may not have preceded the

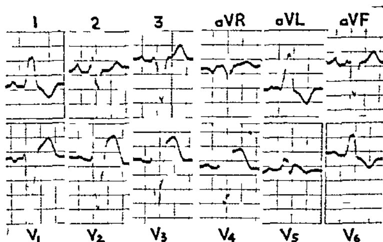


FIG 122 Possible acute anterior infarction with left bundle branch block. The high ST segments in V_{1-4} suggest the diagnosis but these may occur in uncomplicated bundle branch block. Infarction can only be diagnosed with certainty if progressive changes are observed in serial tracings.

Fig 123 Acute anterior infarction with right bundle branch block The Q waves in V_{1-4} together with ST elevation and T inversion in V_{1-5} make the diagnosis Note the evolution in ST T pattern from upper series of precordials to lower series taken a few days apart The standard leads were taken on the day of the lower precordial series

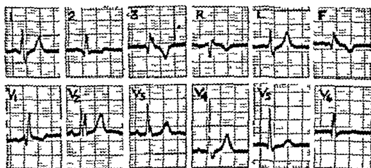


Fig 124 Acute inferior infarction with right bundle branch block Q and T changes are evident in 3 and aVF with reciprocal T wave changes in V_{1-2}

infarction but may have resulted from it indeed the presence of bundle branch block may be considered an integral part of the pattern of septal infarction (28)

Localization of infarction

Much has been written about the scrupulous localization of infarctions Such localization is a good academic exercise but is of relatively little practical value It is of some modest value in prognosis for it is generally agreed that the best prognosis is enjoyed by patients with small anteroseptal lateral or subendocardial infarctions while septal infarctions producing bundle branch block and combined anterior and inferior infarctions carry the worst outlook The most important clinical question however is Has an infarction occurred? , not Where is it?

Localization is mainly based on the previously stated principle that the changes we have learned to associate with the indicative lead (those epitomized in fig 107 page 148) occur in leads facing the damaged surface of the heart Thus if Q ST and T changes are seen in all the precordial leads from V_1 to V_6 we diagnose a **large anterior or**

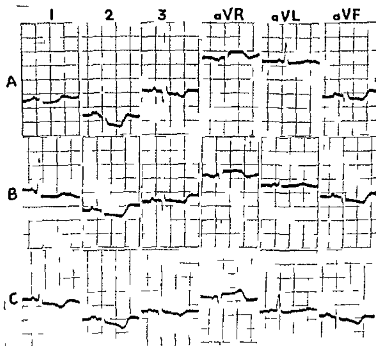


Fig 125 Acute subendocardial infarction Note ST T depression in several leads and the progression of these changes Tracing B was taken 2 days after A C 8 days after B

Infarction without Q waves

Q waves have come to be regarded as the hallowed hallmark of infarction But from what has been said earlier in this chapter concerning their genesis it is apparent that there is nothing sacrosanct about Q waves as such Their real importance is that they represent the replacement of electrical forces directed towards the electrode by oppositely directed forces i e replacement of impulses (dipoles) travelling toward the electrode by impulses travelling away from it This being so loss of R wave with or without gain in depth of S wave might well carry the same significance as a Q wave Indeed in some circumstances this is found to be true Loss of amplitude of QRS complexes over the left ventricle has already been mentioned as an index of infarction in the presence of left bundle branch block

Again reversal of the normal trend in height of R waves in the first three or four precordial leads may be a helpful sign In these leads we have seen that the height of the R wave normally increases from right to left If however these R waves dwindle progressively from right to left it is suspicious of anteroseptal infarction It should be *mentioned that complete loss of R waves (i e QS complexes) in* V_1 through V_4 is not necessarily evidence of anterior infarction and is seen not infrequently in left ventricular hypertrophy (30)

It has been mentioned that in true posterior or infero posterior infarction the only changes may be reciprocal ones observed in the anterior chest leads At times in true posterior or in lateral infarction (19 21) the sole or primary change may be an increase in the height of R wave over the right precordium (V_{3R} V_1 V) Similarly an increased width of the R wave to 0 04 sec or more in V_1 and V may be diagnostic of true posterior infarction (5)

Another infarction pattern of which the most striking feature is *left axis deviation* has been described by Grant (20) Its characteristics are 1) an initial wide R wave (0 04 sec or more) followed by a deep S wave in aVF and 2) marked left axis deviation with prominent terminal R wave in aVR or S wave in lead 1 (fig 126) A careful study with autopsy correlation has shown that patients with this pattern have infarction of the antero lateral wall Grant has also pointed out that *right axis deviation* may sometimes result from infarction of the diaphragmatic wall (5)

It was noted earlier that one of the criteria for diagnosing a subendocardial infarction was the absence of Q waves An explanation for this finding has been recently supplied It has been found that the

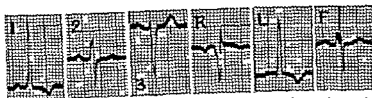


FIG 176 **Anterolateral infarction** pattern described by Grant Note 1) marked left axis deviation 2) prominent R in aVR and 3) wide initial R wave in aVF followed by relatively deep S wave

impulse travels much faster (several thousand mm per sec) through the inner third of the myocardium than it does through the outer two thirds (about 500 mm per sec) In fact it travels so fast that it makes no impression on the depolarization complex (QRS) as recorded by our crude instruments experimentally and in the human heart the QRS remains unaltered when the inner myocardial layers alone are injured (15 16)

It is thus clear that Q waves although they remain the sheet anchor of confident diagnosis are by no means a sine qua non of all infarction patterns

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Another infarction pattern of which the most striking feature is *left axis deviation* has been described by Grant (20) Its characteristics are 1) an initial wide R wave (0 04 sec or more) followed by a deep S wave in aVF and 2) marked left axis deviation with prominent terminal R wave in aVR or S wave in lead 1 (fig 126) A careful study with autopsy correlation has shown that patients with this pattern have infarction of the antero lateral wall Grant has also pointed out that *right axis deviation* may sometimes result from infarction of the diaphragmatic wall (5)

It was noted earlier that one of the criteria for diagnosing a subendocardial infarction was the absence of Q waves An explanation for this finding has been recently supplied It has been found that the

Factors which should be borne in mind as probable causes of missed diagnoses are

1. failure to take serial tracings
2. failure to take additional exploratory chest leads in doubtful cases
3. presence of bundle branch block
4. digitalis action tending to neutralize ST elevations
5. simultaneous infarcts neutralizing each other's patterns

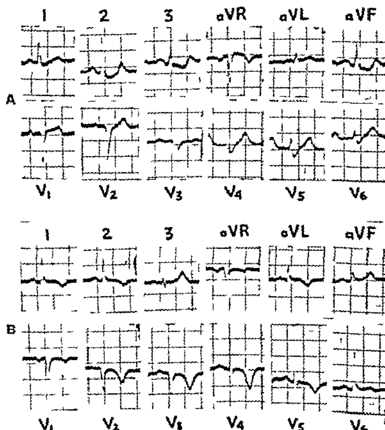


Fig. 128 **Acute anterior infarction** A was taken 8 hours before B Note the rapid evolution of precordial T waves

VALUE OF THE aV LEADS

We have now covered all the various conditions in which the aV leads may be of particular value (29) and it may be helpful to summarize them

1. The **electrical position** of the heart is determined from aVL and aVF (page 38)
2. Occasionally aVL or aVF gives the earliest evidence of **left ventricular strain** (page 52)
3. **A V nodal rhythms** are sometimes recognized most easily or confirmed in aVR and aVF (page 120)
4. Leads aVL and aVF may arbitrate in evaluating the significance of **Q waves in lead 3** (pages 152–3)
5. Occasionally aVL or aVF gives the earliest or best evidence of **myocardial infarction** (pages 150–1)

REFERENCES

ANTERIOR INFARCTION

1. Myers G B Klein H A and Stofer B E Correlation of electrocardiographic and pathologic findings in anteroseptal infarction Am Heart J 1948 36 535
2. Myers G B Klein H A and Hiratzka T Correlation of electrocardiographic and pathologic findings in large anterolateral infarcts Am Heart J 1948 36 838
3. Myers G B Klein H A and Hiratzka T Correlation of electrocardiographic and pathologic findings in anteroposterior infarction Am Heart J 1949 37 205

INFERIOR (FORMERLY "POSTERIOR") INFARCTION

4. Evans W The effect of deep inbreathing on lead III of the electrocardiogram Brit Heart J 1951 13 457
5. Grant R P and Murray R H QRS complex deformity of myocardial infarction in the human subject Am J Med 1954 17 587
6. Myers G B Klein H A and Hiratzka T Correlation of electrocardiographic and pathologic findings in posterior infarction Am Heart J 1949 38 547
7. Myers G B Klein H A and Hiratzka T Correlation of electrocardiographic and pathologic findings in posterolateral infarction *Am Heart J 1949 38 837*
8. Wachtel F W and Teich E M Tall precordial T waves as the earliest sign of diaphragmatic wall infarction Am Heart J 1956 51 91
9. Weisbart M H and Simonson E The diagnostic accuracy of Q and *related electrocardiographic items for the detection of patients with* posterior wall myocardial infarction Am Heart J 1955 50 62

10 Yu P N G and Blake T M The significance of QaVF in the diagnosis of posterior infarction Am Heart J 1950 40 545

INFARCTION COMPLICATED BY BBB

11 Dressler W Roesler H and Schwager A The electrocardiographic signs of myocardial infarction in the presence of bundle branch block I Myocardial infarction with left bundle branch block Am Heart J 1950 39 217
12 Dressler W Roesler H and Schwager A The electrocardiographic signs of myocardial infarction in the presence of bundle branch block II Myocardial infarction with right bundle branch block Am Heart J 1950 39 544
13 Kennamer R and Prinzmetal M Myocardial infarction complicated by left bundle branch block Am Heart J 1956 51 78

SUBENDOCARDIAL INFARCTION

14 Levine H D and Ford R V Subendocardial infarction report of six cases and critical survey of the literature Circulation 1950 1 246
15 Massumi R A et al Studies on the mechanism of ventricular activity XVI Activation of the human ventricle Am J Med 1955 19 832
16 Prinzmetal M et al Studies on the mechanism of ventricular activity VI The depolarization complex in pure subendocardial infarction role of the subendocardial region in the normal electrocardiogram Am J Med 1954 16 469
17 Pruitt R D Klakeg C H and Chapin L E Certain clinical states and pathologic changes associated with deeply inverted T waves in the precordial electrocardiogram Circulation 1955 11 517
18 Yu P N G and Stewart J M Subendocardial myocardial infarction with special reference to the electrocardiographic changes Am Heart J 1950 39 862

LATERAL INFARCTION

19 Dunn W J Edwards J E and Pruitt R D The electrocardiogram in infarction of the lateral wall of the left ventricle a clinicopathologic study Circulation 1956 14 540
20 Grant R P Left axis deviation An electrocardiographic pathologic correlation study Circulation 1956 14 233
21 Levy L et al Prominent R wave and shallow S wave in lead V as a result of lateral myocardial infarction Am Heart J 1950 40 447
22 Myers G B Klein H A and Stofer B E Correlation of electrocardiographic and pathologic findings in lateral infarction 1949 37 374

MISCELLANEOUS

23 Bayley R H LaDue J S and York D J Electrocardiographic changes (local ventricular ischemia and injury) produced in the dog by temporary occlusion of a coronary artery showing a new stage in the evolution of myocardial infarction Am Heart J 1944 27 164

24 Dressler W and Roesler H The diagnostic value of the pattern T lower than T_3 ($T_1 < T_3$) compared with the information yielded by multiple chest leads in myocardial infarction Am Heart J 1948 36 115
25 Dressler W and Roesler H High T waves in the earliest stage of myocardial infarction Am Heart J 1947 34 627
26 East T and Oram S The cardiogram in ventricular aneurysm following cardiac infarction Brit Heart J 1952 14 12
27 Freundlich J The diagnostic significance of tall upright T waves in the chest lead Am Heart J 1956 52 749
28 Osher H L and Wolff L The diagnosis of infarction of the interventricular septum Am Heart J 1953 45 429
29 Sokolow M The clinical value of the unipolar extremity (aV) leads Ann Int Med 1951 34 921
30 Surawicz B et al QS and QR pattern in leads V_3 and V_4 in absence of myocardial infarction electrocardiographic and vectorcardiographic study Circulation 1956 12 391

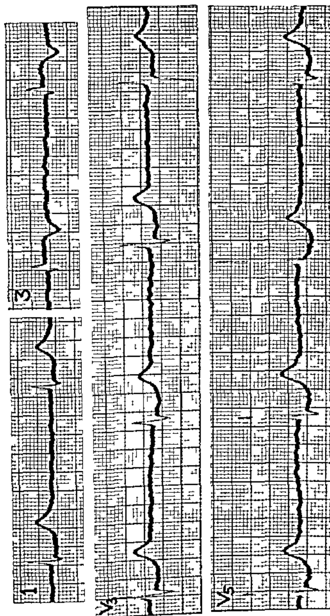


Fig G For interpretation see p 213

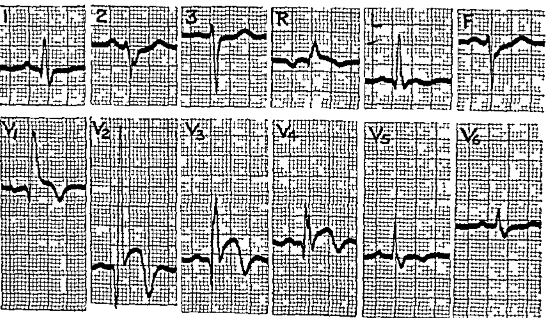


Fig II For interpretation see p 213

13

Coronary Insufficiency and Related Matters

Coronary insufficiency may be suspected when T waves are flattened or inverted in many leads with or without accompanying ST depression (figs 129 and 130) The pattern of left ventricular strain obviously falls into this category and no doubt the strain pattern is at least partly the result of myocardial ischemia It should be remembered however that a number of other conditions cause ST T changes which may readily be confused with those of coronary disease These will be dealt with later in this chapter

An ST T pattern particularly suggestive of coronary insufficiency is a horizontal ST segment (also known as plane depression (1)) making a sharp angle with the proximal shoulder of the still normally upright T wave (figs 129 and 130 C) Normally the ST segment and T wave should merge smoothly and imperceptibly

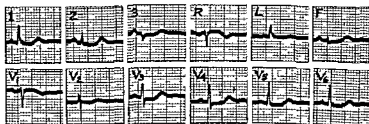


Fig 129 Coronary insufficiency Note horizontally depressed ST segments in many leads

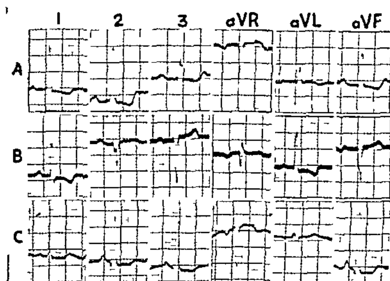


FIG 130 Coronary insufficiency ST T depression in many leads not conforming to any specific pattern note the sharp angled junction between ST segments and the still upright T waves in several leads in tracing C

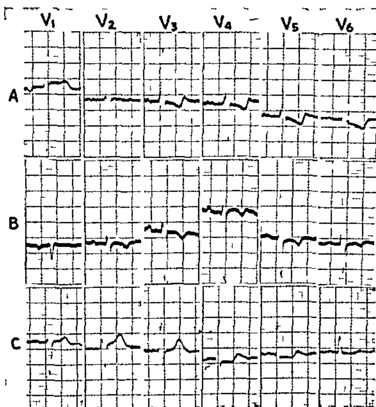


Fig 130 (continued)

At times the most striking or only evidence of coronary insufficienc is inverted U waves (fig 131)

Another important sign of coronary insufficiency is post extr systolic T wave inversion (2 3) The T wave of the sinus beat follow ing the premature beat is distorted usually inverted sometimes th change is accompanied by abnormal lengthening of the Q T interv (fig 132) Levine has called this the poor man s exercise test' that the change is included in the initial tracing and so obviates th need and expense of a subsequent exercise test

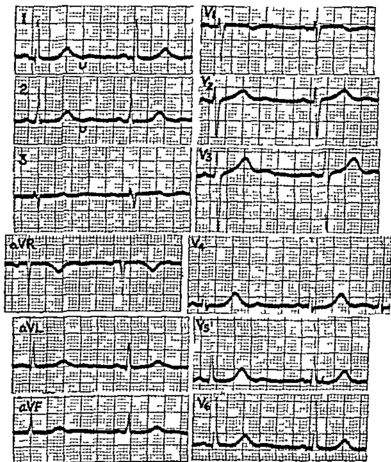


Fig 131 Coronary insufficiency ST segments are rather horizontal but the striking abnormality is U wave inversion most pronounced in leads 1 and 2

3

FIG 132 **Post extrasystoli T w r e changes** After two sinus beats three of the next four beats are ve toles from diff rent foci (multi form extrasy toles) Followii tole the next sinus beat shows marked increa e in T wave ii a ed Q T duration (0 38 sec) the next beat shows omewl ver ion and Q T duration has le sened (0 3? s c)

FIG 133 **Coronary insufficiency** Lead 2 take 1 luring an attack of chest pain which la te 1 le s than a minute Top left corner trip taken shortly before pain began spontaneou ly figure on ubsequent strip represent number of secon ls after on t of pain N tice marked progre ive ST T depression and appearance of frequent ectopic ventricular beat At end of two minutes pat tern has returned to normal

In some cases of coronary insufficiency abnormalities in the electro cardiogram occur only *during* an attack of pain (fig 133) and in others the inadequacy of the coronary circulation can only be dem onstrated after exercise (4) or after a period of anoxia This is the basis for the various exercise and anoxia te ts The electrocardiogram being normal while the patient is at rest he is subjected to exercise or to an atmosphere deficient in oxygen and tracings are again taken

The appearance in these circumstances of flattened or inverted T waves and depressions of the ST segments of 1 mm or so indicates coronary insufficiency (fig 134) In the healthy heart the changes which normally follow exercise are tachycardia increased height of P wave decreased P R interval slight ST depression (not more than 0 5 mm) and increased height of T waves

Finally, it should be clearly appreciated that the presence of coronary disease is not excluded by a normal tracing The diagnosis of

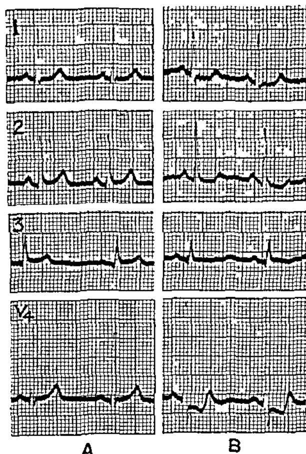


Fig 134 **Positive exercise test** A Control tracing before exercise within normal limits B Two minutes after exercise striking ST depression in V_4 with lesser ST T changes in 1 and 2

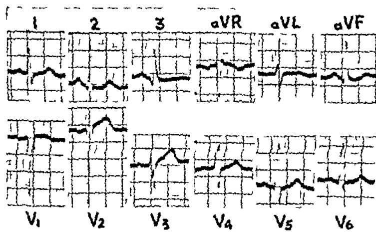


FIG 135 Normal tracing in a patient who has suffered from angina of effort for eight years

angina is made primarily from the history Figure 135 is the tracing from a 53 year old man who had a clear history of angina of effort for 8 years The electrocardiogram is within normal limits

ELECTROCARDIOGRAPHOGENIC DISEASE

It should be emphasized that abnormalities in the electrocardiogram do not necessarily indicate cardiac disease much less coronary disease (10) When deviations from the normal especially those affecting the S-T segments and T waves are encountered in the middle aged and elderly they are often much too glibly interpreted as coronary insufficiency Statistically such inferences are no doubt often right but the habit is bad practice and is scientifically unsound too many people are limping their ways through life maimed by the unkind cuts of electrocardiographic interpretation (15) The following facts should always be remembered before the cardiac or coronary label is attached

1) **The range of normal is wide and its limits cannot be satisfactorily defined** (19) Changes well outside the accepted range are undoubtedly at times normal variants Examples of this are the

The appearance in these circumstances of flattened or inverted
waves and depressions of the ST segments of 1 mm or so, indicate
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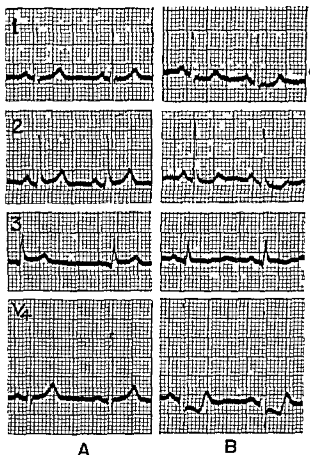


FIG 134 Positive exercise test A Control tracing before exercise within normal limit B Two minutes after exercise striking ST depression in V_4 with lesser ST T changes in 1 and 2

thenia gravis all may produce changes in the tracing indicative of myocardial involvement and quite indistinguishable from some of the alterations resulting from coronary disease

Therefore in assessing the tracing that does not conform with our accepted standards we should remember the whole array of common and uncommon possibilities and we should ask ourselves three questions 1) Could this be a normal variant? 2) Could these abnormalities be due to extracardiac factors physiological or pathological? and 3) Could these changes be due to heart disease other than coronary?

The danger of attributing changes of the first and second category to heart disease is that the patient is branded as a cardiac The danger of labelling the third group as coronary is that the physician in charge of the case may be thereby blinded to the true nature of the cardiac involvement and of the underlying primary disease In the end we should often be content to state that the pattern is abnormal but non specific We should also certainly be at pains to spread the gospel that AN ABNORMAL TRACING DOES NOT NECESSARILY MEAN AN ABNORMAL HEART

REFERENCES

CORONARY INSUFFICIENCY

1 Evans W and McRae C The lesser electrocardiographic signs of cardiac pain Brit Heart J 1952 14 429
2 Levine H D Lown B and Streeper R B The clinical significance of post extra systolic T wave changes Circulation 1952 6 538
3 Mann R H and Burchell H B The sign of T wave inversion in sinus beats following ventricular extrasystoles Am Heart J 1954 47 504
4 Scherf D and Schaffer A I The electrocardiographic exercise test Am Heart J 195 43 92

ELECTROCARDIOGRAPHIC HOGENIC DISEASE

5 Beckner C L and Winsor T Cardiovascular adaptations to prolonged physical effort Circulation 1954 9 835
6 Burch G E Meyers R and Abildskov J A A new electrocardiographic pattern observed in cerebrovascular accident Circulation 1954 9 719
7 de la Chapelle C E and Kossmann C I Myocarditis Circulation 1954 10 74
8 Dowling C V and Hellerstein H K Factors influencing the T wave of the electrocardiogram II Effect of drinking iced water Am Heart J 1951 41 58
9 Grusin H Peculiarities of the African electrocardiogram and the changes observed in serial studies Circulation 1954 9 860
10 Levine H D Non specificity of the electrocardiogram associated with coronary artery disease Am J Med 1953 15 344

11 Littmann D Persistence of the juvenile pattern in the precordial leads of healthy adult Negroes with report of electrocardiographic survey on three hundred Negro and two hundred white subjects Am Heart J 1946 32 370
12 Magendantz H and Shortsleeve J Electrocardiographic abnormalities in patients exhibiting anxiety Am Heart J 1951 42 849
13 Manning G W Electrocardiography in the selection of Royal Canadian Air Force Aircrew Circulation 1954 10 401
14 Mitchell J H and Shapiro A I The relationship of adrenalin and T wave changes in the anxiety state Am Heart J 1954 48 323
15 Prinzmetal M et al Clinical implications of errors in electrocardiographic interpretation Heart disease of electrocardiographic origin J Am Med Assoc 1956 161 138
16 Rochlin I and Edwards W L J The misinterpretation of electrocardiograms with postprandial T wave inversion Circulation 1954 10 843
17 Scherf D and Dix J H The effects of posture on A V conduction Am Heart J 1952 43 494
18 Silverman J J and Goodman R D Extraordinary alteration of P R interval in neurocirculatory asthenia Am Heart J 1951 41 155
19 Simonson E Editorial Principles for determination of electrocardiographic normal standards Am Heart J 1956 52 163
20 Simonson E and Keys A The effect of an ordinary meal on the electrocardiogram Normal standards in middle aged men and women Circulation 1950 1 1000
21 Wasserburger R H Siebecker K L and Lewis W C The effect of hyperventilation on the normal adult electrocardiogram Circulation 1956 13 850

Review Tracing

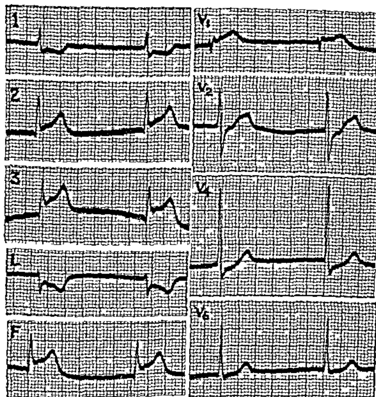


Fig I

For interpretation see p 213

Review Tracing

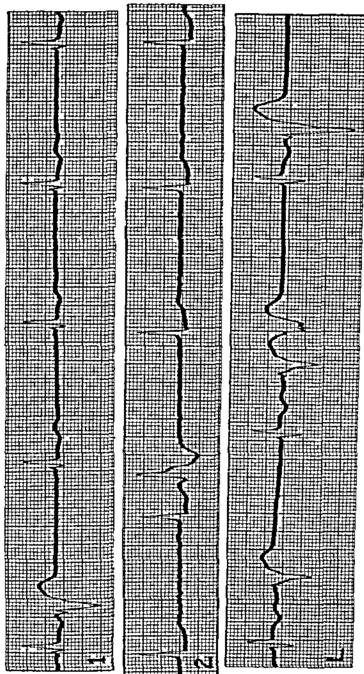


Fig J For interpretation see p 214

14

Miscellaneous Conditions

This chapter is not an exhaustive section illustrating all the innumerable circumstances in which the electrocardiogram deviates from normal. It includes only those conditions in which the tracing is or may be of diagnostic value.

VALVULAR LESIONS

The electrocardiogram plays only a small part in the diagnosis of valvular lesions. *Mitral stenosis is the only one which may claim* anything like a specific pattern (1). The **P mitrale** pattern, consisting of wide notched P waves in leads 1 and 2 with flat, biphasic or inverted P waves in 3, is frequently found (fig. 137). The combination

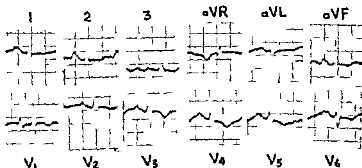


Fig. 137 Mitral stenosis. Note P mitrale pattern, right axis deviation and tall R in V1 with deep S in V6.

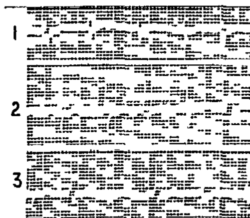


Fig 138 Mitral stenosis Note atrial fibrillation right axis deviation tall R or R waves in $CF_{1,2}$ and deep S waves as far to the left as CF_4

of right axis deviation (with or without right ventricular strain) and the P mitrale pattern or atrial fibrillation is strongly suggestive of mitral stenosis (fig 138) It has been stated that the combination of right axis deviation with atrial fibrillation in a patient under forty is practically diagnostic of mitral stenosis This combination however is sometimes found in thyrotoxicosis and in atrial septal defect

The effect of other valvular lesions can be predicted from the known mechanical effects on the heart Aortic regurgitation or stenosis for example predominantly affect the left ventricle producing left ventricular hypertrophy and strain signs of which may appear in the tracing

ACUTE COR PULMONALE

The pattern of acute cor pulmonale is not often seen It develops rapidly and if the patient survives usually reverts to normal within hours to a day or two Probably many are missed because the patient dies or the tracing has reverted to normal before the cardiogram is taken

The pattern develops within a few minutes of a massive pulmonary embolism (3 5) or may develop in the course of many other conditions producing acute cor pulmonale (4) Its greatest importance diagnos

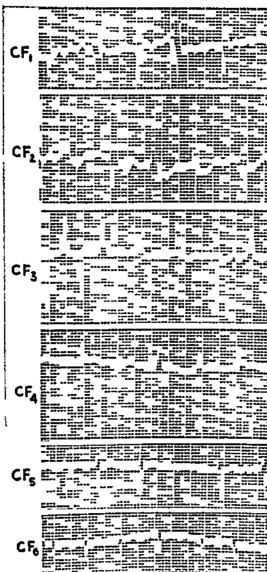


FIG 138 (continued)

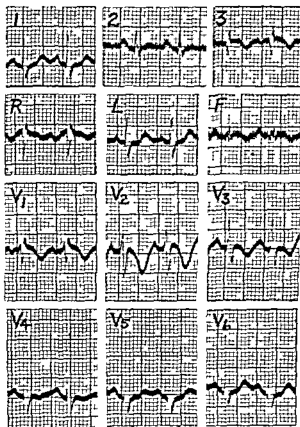


FIG 139 **Acute cor pulmonale** Note Q T_3 pattern consistent with inferior infarction marked T wave inversion over right precordium (V_1-V_4) prominent S wave in 1 and 2

tically is that its pattern somewhat resembles that of inferior myocardial infarction and as the clinical picture also may well be confused with myocardial infarction the distinction is a difficult one. In the typical case, a Q wave develops in lead 3 and the ST segment becomes elevated with shallow inversion of the T wave. Meanwhile lead 1 has developed somewhat reciprocal changes: an S wave appears (indicating a not surprising tendency to develop right axis deviation), the ST segment is depressed while the T remains upright. All these changes are compatible with inferior infarction. Lead 2, however, tends to follow lead 1 and shows no Q wave but an S wave, a slightly depressed ST segment and an upright T wave, whereas in inferior infarction lead 2 tends to follow lead 3 with a Q, an elevated ST and inverted T.

In the precordial leads elevated ST segments and inverted T waves are sometimes seen over the right ventricle while S waves may become more prominent over the left ventricle (indications of right ventricular dilatation). Transient right bundle branch block may appear. Many of these changes are to be seen in figure 139.

The differences between this pattern and that of inferior infarction may thus be summarized as follows:

1. lead 2 tends to follow lead 1 rather than 3
2. the changes are fleeting, evolving and receding in a matter of hours rather than weeks or months
3. ST-T deviations in limb leads are slight, whereas they may be major in inferior infarction, and in right precordial leads they resemble the anteroseptal rather than the inferior infarction pattern.

CHRONIC COR PULMONALE

Chronic cor pulmonale most often seen in emphysema is characterized by right axis deviation and sometimes the pattern of right ventricular strain Strain on the right atrium is manifested by the **P pulmonale** pattern (fig 140 also fig 11 page 19) consisting of a low P in lead 1 with tall pointed P waves in 2 3 and aVF The P waves *in right precordial leads are usually also pointed but inverted* or are biphasic with a distinct intrinsicoid deflection Low voltage is not infrequently present and T_1 is often of lower voltage than T_3

Frequently instead of the full blown pattern of right ventricular hypertrophy and strain with tall R waves in V_1 an intermediate pattern is seen with deep S waves across the precordium from V_1 to V_6 (fig 140) The Q T interval in cor pulmonale unlike that in other forms of heart failure is not prolonged (2) This may at times be a helpful differential point

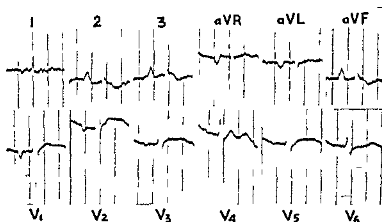


FIG 140 **Chronic cor pulmonale** Note P pulmonale pattern vertical heart and deep S waves across the precordium (clockwise rotation)

Salient features of chronic cor pulmonale 1 Right axis deviation 2 Right ventricular strain pattern or simply marked clockwise rotation 3 P pulmonale pattern 4 Often low voltage QRS and T_1 lower than T_3

ACUTE PERICARDITIS

In acute pericarditis from whatever cause the characteristic finding is an elevation of ST segments with upward concavity in all three standard leads and often in other leads as well The T wave remains upright at first except in lead 3 where it may be inverted Lead 3 is also often an exception in the shape of its ST segment which may present an upward convexity These changes characterize the first or **ST stage** of acute pericarditis (fig 141)

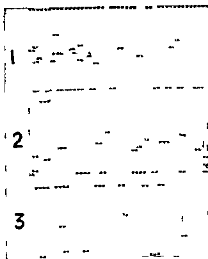


Fig 141 Acute pericarditis (in this case an *acute hemopericardium*) Note ST elevation in all three standard leads with upward concavity in leads 1 and 2

The second stage or **T stage** presents T wave inversion in the three standard leads and often in other leads At this stage the ST segments have returned to the isoelectric level During both stages low voltage is a common finding In the average case of acute pericarditis resolving in the course of three or four weeks these stages each last for about ten days to two weeks

The four most striking differences between acute pericarditis and acute infarction are tabulated

	ACUTE PERICARDITIS	ACUTE INFARCTION
ST reciprocity (between 1 and 3)	Absent Elevation in both 1 and 3	Present Elevated in one depressed in the other
ST shape	Concave upwards	Convex upwards
Q waves	Absent	Present
Period of evolution	Few weeks	Months

The changes in pericarditis are probably due to two causes

1 short circuiting of impulses by pericardial fluid or thickened pericardium causes the low voltage
2 spread of the inflammation to the immediately subjacent layer of myocardium (i e subepicardial myocarditis) accounts for the ST and T wave changes

CHRONIC CONSTRICTIVE PERICARDITIS

In the chronic constrictive or adhesive types of pericarditis changes in the tracing are relatively fixed and non progressive They are not unlike the findings in the T stage of the acute disease two of the most characteristic features being low voltage and inverted T waves Flat or inverted T waves are present in all cases abnormal P waves in about three quarters of the cases and low voltage in over half (6) Such changes are found in the three standard leads and in all or many of the other leads It is of practical importance to note the degree of inversion of the T waves for the depth of inversion is usually proportional to the degree of pericardial adherence to the myocardium (7) deep T waves are associated with intimate adherence which

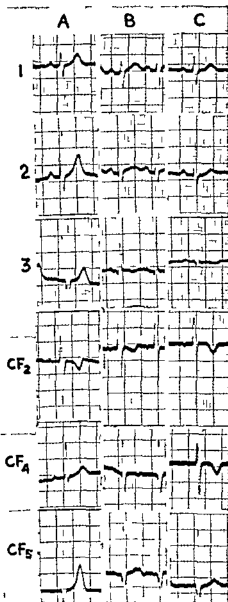


FIG 14? Acute pericarditis (in a child with rheumatic fever)

A Tracing taken before pericarditis developed B Five weeks later at height of pericarditis C Two days later Note evolution of T wave changes especially in leads 2 3 CF and CF₅ In tracing B note the notched but still upright T wave in CF

makes surgical stripping difficult or impossible whereas flat or barely inverted T waves usually indicate a relatively easy surgical undertaking

Atrial fibrillation is persistently present in over a third of the cases (6)

One other characteristic which deserves passing mention is that the axis of the heart does not alter as it does in the normal when the patient turns from one side to the other for being bound by adhesions it is not free to swing from side to side with change in position This electrocardiographic feature corresponds with the clinical finding of a fixed apex beat

Salient features of chronic pericarditis

1 Low voltage
2 Flat or inverted T waves
3 Fixed axis
4 Possible P mitrale pattern or atrial fibrillation

THE HEART IN CHILDHOOD AND CONGENITAL LESIONS

Several points are of importance in interpreting the electrocardiogram in children First and foremost variations in the normal are more diverse than they are in adult tracings so that one should be even more careful in declaring a youthful tracing abnormal than that of an adult The rate is relatively faster and the P R and QRS intervals relatively shorter in childhood

At birth the right ventricle is larger than the left and this leads to a different balance of power Apart from the common occurrence of right axis deviation tall R waves are frequently seen in precordial leads to the right of the precordium with deep S waves present over the left ventricle Thus a pattern reminiscent of right ventricular hypertrophy or right bundle branch block in the adult may be a perfectly normal finding in the child

A further important point to remember is that T waves may be normally inverted further to the left of the precordium in the child (see page 23)

There are only two specifically diagnostic patterns in congenital heart disease both very rare

1 When P QRS and T are all inverted in lead 1 so that the lead presents a mirror image of the normal and leads aVR and aVL are also transposed dextrocardia with complete situs inversus may be diagnosed It is important to realize that this pattern is indistinguishable from that produced when the arm electrodes are reversed (fig 143) This technical error is obviously much more frequently encountered than the congenital anomaly

2 The pattern of anterior myocardial infarction in infancy makes the diagnosis of anomalous left coronary originating from the pulmonary artery

Non specific patterns are far more common Of these the commonest is that of **right ventricular strain** which may be found in a variety of congenital lesions including pure pulmonary stenosis atrial septal defect with or without mitral stenosis pulmonary stenosis with atrial or small ventricular septal defect the tetralogy of Fallot Eisenmenger s complex and transposition of the great vessels

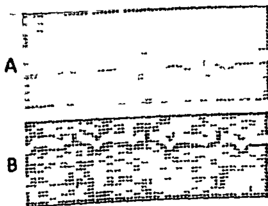


Fig 143 A Lead 1 in dextrocardia B Lead 1 from a normal heart with the arm electrodes reversed

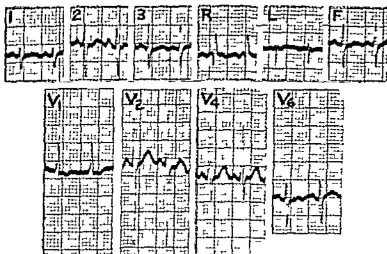


FIG 144 **Congenital heart disease** From a cyanotic baby one day old Standard limb lead show **Katz Wachtel phenomenon** and precordial leads show pattern of right ventricular hypertrophy

Another commonly encountered pattern is the **Katz Wachtel phenomenon** which consists in the presence of relatively equiphasic QRS complexes in two or three of the standard limb leads (fig 144) This pattern may be found in several congenital defects including many of the above and it may also occur in other abnormalities such as isolated ventricular septal defect and single ventricle

Left ventricular strain (8) is much less common but may develop in a number of lesions including coarctation of the aorta aortic stenosis tricuspid atresia and severe degrees of patent ductus arteriosus Notice that tricuspid atresia is the only cyanotic form of congenital heart disease associated with left axis deviation

Finally the tracing may remain entirely normal especially in milder grades of patent ductus, small septal defects and coarctation of the aorta At the other extreme a large atrial septal defect besides producing marked right ventricular hypertrophy and strain may be complicated by a number of other electrocardiographic abnormalities including right bundle branch block A V block and atrial fibrillation

DIGITALIS AND THE ELECTROCARDIOGRAM

Digitalis is to the electrocardiogram what syphilis *was* to medicine—the great imitator It can mimic heart disease and it can cause all types of block and all manner of arrhythmias It is of great importance to the clinician to appreciate the significance of the various changes produced by digitalis and so these will be considered in some detail

It is most convenient to divide the effects of digitalis into four groups

1 *ST T changes* digitalis causes depression of the ST segments with flattening and inversion of T waves At the same time the relative Q T duration is shortened in contrast to quinidine effect (see below) The shape of the depressed segment *is often characteristic—it is sagging with its concavity upwards* and has been said to look as though a finger had been hooked over it to drag it down (figs 145 and 146) These are not indications of digitalis *intoxication* but rather of simple digitalis *effect* They may be anticipated in most patients who are approaching adequate digitalization and are not necessarily an indication for reducing dosage These changes occur in animals with approximately 25 per cent of the lethal dose

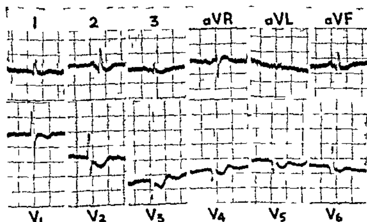


Fig 145 Digitalis effect Note sagging ST segments in most leads

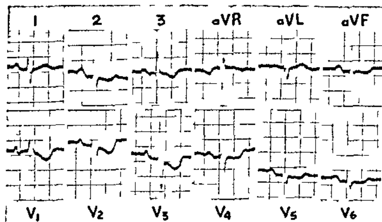


FIG 146 Digitalis effect Note sagging ST segments in most leads especially in V1 2

It should be noted that ST depression and inversion of T waves usually occur only in those leads with tall R waves It is claimed that such displacement of ST segments and T waves in the direction opposite to the main QRS deflection means a uniform therapeutic action on the myocardium but that depression of ST and inversion of T also in leads with mainly negative QRS complexes indicates that the drug is causing relative coronary insufficiency in the sub endocardial muscle layers and is therefore an indication to reduce the dose

2 *Premature ventricular beats* often proceeding to *bigeminal rhythm* These are of course arrhythmias and as such should be classified with the other arrhythmias below But they are so much more often encountered than any other ar rhythmia and are therefore so suggestive of digitalis action, that they deserve a category to themselves They are a sign of definite toxicity and are an indication to revise dosage In animals they appear after approximately 70 per cent of the lethal dose has been administered

3 *Arrhythmias* Almost every arrhythmia has been reported to complicate digitalis administration Premature ventricu lar beats have already been stated to be the commonest but their kindred more serious rhythms ventricular tachy

cardia and ventricular fibrillation have occasionally occurred Atrial arrhythmias may also be produced and premature beats tachycardia and fibrillation have all been attributed to digitalis action Digitoxin seems particularly likely to induce arrhythmic tachycardias A V dissociation with interference is sometimes produced by digitalis

4 *Blocks* Sinu atrial atrio ventricular and intraventricular blocks have all followed digitalis administration S A block may induce the onset of A V nodal rhythm Simple lengthening of the P R interval is common and partly results from vagal stimulation (fig 98 B page 134) In animals it is induced by about 50 per cent of the lethal do e Higher grades of A V block are not infrequently seen Complete A V block may develop without any warning symptoms in a patient maintained on digitoxin such blo k in animals indicates about 80 per cent of the lethal dose Prolongation of the QRS interval occurs rarely in digitalis intoxication

Slowing of the heart in sinus rhythm is not due to heart block but to enhanced vagal effect on the S A node In atrial fibrillation slowing of the ventricle is the desirable result of A V block

QUINIDINE

In general quinidine causes qualitatively similar but quantitatively different changes in the tracing The noticeable exception to this general statement is its effect on the Q T interval which it regularly lengthens in contrast to digitalis effect It is le s likely to cause lengthening of the P R interval but much more likely to prolong the QRS

Its influences may be summarized as follows

1 *ST T changes* T waves become depressed widened notched and finally inverted Meanwhile the Q T interval lengthens (fig 147) The ST segment is less likely to become depressed than with digitalis administration

2 *Blocks* of all types can occur S A block may produce fatal atrial standstill Prolongation of the QRS is frequently seen and is important to the therapist if this interval increases during treatment by 25–50 per cent it is an indication to discontinue the drug

3 *Ventricular ectopic rhythms* are occasionally produced

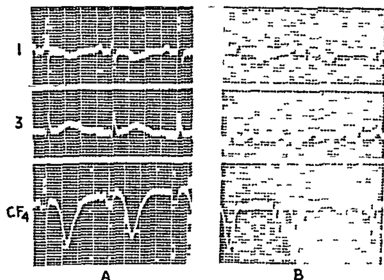


FIG 147 **Quinidine effect** Note the markedly prolonged Q T interval in A of about 0 54 seconds (upper limit of normal in a male at this rate 72 is 0 395 seconds) quinidine effect has pulled apart the limbs of the inverted coronary T waves in CF In B the Q T interval has returned to normal (0 36 seconds) Quinidine had been administered for two weeks before tracing A it was discontinued on the day of this tracing and tracing B was taken one week later

MYXEDEMA

The diagnosis of myxedema should certainly never depend upon electrocardiographic changes though it may be suspected when flattening or shallow inversion of many T waves is seen without comparable ST displacement (fig 148) Its characteristics are three

1 low voltage
2 sinus bradycardia
3 low to inverted T waves in all or many leads



Fig 148 Myxedema Tracing A was taken before treatment note shallow inversion of T waves in many leads B was taken after ten weeks of treatment with thyroid extract the previously inverted T waves are now upright

HYPOKALEMIA

The electrocardiogram may be of great value in the diagnosis of this not uncommon and dangerous situation A significant potassium deficit may be encountered in many metabolic disorders including

cirrhosis of the liver diabetic coma after vigorous treatment hypochloremic alkalosis from whatever cause (vomiting mercurial diuresis, etc) and in situations where excessvie amounts of corticosteroids are being secreted (Cushing s syndrome primary aldosteronism) or administered It has been demonstrated that the typical signs of potassium lack in the tracing may appear when the serum potassium is within normal limits and conversely that the tracing may be normal and show no evidence of potassium deficiency when hypokalemia is chemically proven As it is the heart that is most dangerously affected by too much or too little potassium it may well be that the electrocardiogram is the most sensitive indicator of the immediate threat to life Furthermore an electrocardiogram can often be taken when facilities for chemical determinations or flame photometry are not available It is therefore worth while to know the changes that a potassium deficit can initiate (figs 149 and 150) These usually occur in the following sequence

1 Apparent prolongation of the Q T interval (This appears to be due to flattening and widening of the T wave but more careful analysis has shown that it is due to a lower T wave and taller U wave merging to form a continuous wide wave this early stage is shown in figure 150 A)
2 T wave inversion
3 Sagging ST segment and finally a low "take off" of this segment

The fully developed pattern is seen in figure 150 B These changes rapidly revert to normal with administration of potassium salts



FIG 149 Hypokalemia Tracing in A shows well marked signs of potassium deficiency—low ST take off with prominent U waves giving appearance of prolonged Q T interval B Four days later C Ten days later tracing has returned to normal

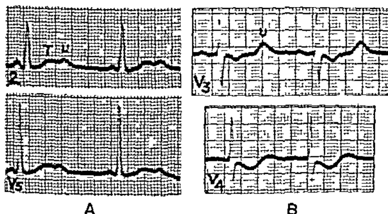


Fig 150 Hypokalemia Tracings A and B are from different patients A shows early changes of hypokalemia with prominent U wave merging to form continuous undulating wave with T wave B shows changes of advanced hypokalemia (1.8 mEq /liter) in a patient with cirrhosis note ST T depression with very prominent U waves in V3

HYPOCALCEMIA

Calcium deficiency produces a prolonged Q T interval This lengthening is effected through elongation of the ST segment the T wave remaining relatively normal (fig 151) terminal T wave inversion however occurs in some leads in about a third of the cases

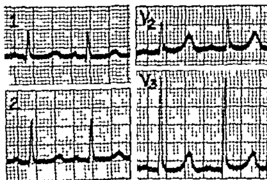


Fig 151 Hypocalcemia Note the prolonged Q T interval in an otherwise normal tracing Q T = 0 40 sec (upper limit of normal for this rate and sex is 0 3 sec) Patient s serum calcium was 7 0 mg /100 ml other electrolytes being normal

HYPERKALEMIA

The earliest sign of potassium intoxication is the appearance of tall thin T waves (fig 152) Later the P R interval becomes prolonged the ST segment becomes depressed and the QRS interval lengthens Finally the P waves disappear and the QRS widens further (fig 153) until ventricular fibrillation closes the picture

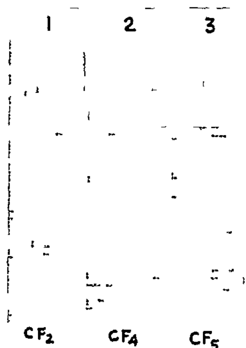


FIG 152 Hyperkalemia in a patient dying in uremia Note extremely tall thin T waves in precordial leads

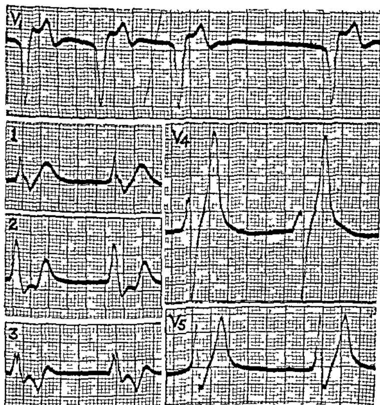


Fig 153 **Hyperkalemia** This tracing shows evidence of advanced potassium intoxication tall peaked T waves absent P waves widened QRS complexes and irregular rhythm From a patient with serum level of 8 1 mEq /liter

ELECTRICAL ALTERNANS

This abnormality is readily recognized by the alternating amplitude of QRS complexes in any or all leads (figs 154 and 155) This is much less common than but has the same prognostic significance as its mechanical counterpart *pulsus alternans*

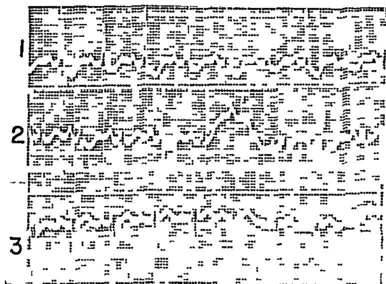


Fig 154 Electrical alternans Note alternating amplitude of QRS complexes in leads 1 and 2 Patient had malignant hypertension and pulsus alternans

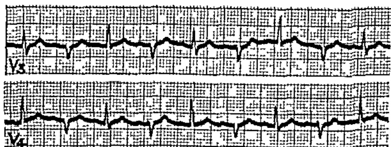


Fig 155 Electrical alternans Note alternating direction of QRS complexes

LOW VOLTAGE WITH INVERTED T WAVES ✓

It is opportune to review the several conditions which can cause low voltage QRS with inverted T waves in all or most leads

1 Any diffuse myocardial involvement
   a diffuse coronary disease
   b heart failure treated with digitalis
   c myxedema
   d amyloidosis
2 Pericarditis
   a acute (T stage)
   b chronic constrictive

ST T DEPRESSION ✓

When ST segments are depressed and T waves flat to inverted in many leads one should think of

1 digitalis effect
2 diffuse coronary disease
3 left ventricular strain
4 combined anterior and inferior infarction (antero inferior infarction)
5 subendocardial infarction
6 hypokalemia

As well as the above causes of ST segment and T wave changes the many factors that can influence these labile members of the electrocardiogram (page 182) should be constantly borne in mind

REFERENCES

MITRAL DISEASE

1 Fraser H R L and Turner R Electrocardiography in mitral valvular disease Brit Heart J 1955 17 459

COR PULMONALE

2 Alexander J K et al The Q T interval in chronic cor pulmonale Circulation 1951 3 733
3 Eliaser M and Giansiracusa F The electrocardiographic diagnosis of acute cor pulmonale Am Heart J 1952 43 533
4 Mack I Harris R and Katz L N Acute cor pulmonale in the absence of pulmonary embolism Am Heart J 1950 39 664
5 Phillips E and Levine H D A critical evaluation of extremity and precordial electrocardiography in acute cor pulmonale Am Heart J 1950 39 205

Fig J page 186 1) Atrial fibrillation 2) Complete A V block with idioventricular rhythm at rate about 38 3) Multiform ventricular premature beats

Fig K page 211 *1) P waves are inverted in 2 3 and aVF upright in aVR* this is therefore an ectopic atrial (? coronary sinus) rhythm 2) 2 to 1 A V block (atrial rate 84 ventricular 42) 3) Left ventricular hypertrophy and strain 4) Digitalis effect

Fig L page 212 1) Sinus bradycardia with arrhythmia rate 43–55 2) Intra atrial block (P wave duration = 0.14 sec) with P mitrale note left axis deviation of P waves with right axis deviation of QRS—this combination is highly suggestive of mitral stenosis From a patient with rheumatic heart disease

Fig M page 212 1) ST T pattern suggests left ventricular strain 2) The third beat in each lead is a premature ventricular beat 3) Following the premature beats retrograde conduction to the atria occurs (retrograde P waves are seen deforming the ST segments) 4) Post extrasystolic T wave changes (increase in depth of T wave inversion) are noted in the cycles following the premature beats From a patient with severe hypertension

INDEX TO ILLUSTRATIVE ELECTROCARDIOGRAMS

(Figure number is given first followed by page in parenthesis)

GENERAL INDEX